SPECTRUM OF PERFORATION PERITONITIS AND EVALUATING THE EFFICACY OF MANNHEIM PERITONITIS INDEX IN PREDICTING THE PROGNOSIS

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CERTIFICATE

This is to certify that this dissertation entitled **"SPECTRUM OF PERFORATION PERITONITIS AND EVALUATING THE EFFICACY OF MANNHEIM PERITONITIS INDEX IN PREDICTING THE PROGNOSIS"** is a record of bonafide research work done by Dr.Prasanna.C.M., under my guidance and supervision in the Department of General Surgery, PSG Institute of Medical Sciences and Research, Coimbatore – 641004.

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DECLARATION

I, Dr.Prasanna.C.M., solemnly declare that this dissertation **"SPECTRUM OF PERFORATION PERITONITIS AND EVALUATING THE EFFICACY OF MANNHEIM PERITONITIS INDEX IN PREDICTING THE PROGNOSIS"** is a bonafide record of work done by me in the Department of General Surgery, PSG institute of Medical Sciences & Research, Coimbatore, under the guidance of Dr.Mahendran.M.G., Professor of Surgery.

This dissertation is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai, in partial fulfilment of the University regulations for the award of MS Degree (General Surgery) Branch-I, Examination to be held in April 2014.

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SPECTRUM OF PERFORATION PERITONITIS AND EVALUATING THE EFFICACY OF MANNHEIM PERITONITIS INDEX IN PREDICTING THE PROGNOSIS

ABSTRACT

Introduction:

Perforation peritonitis has been found to be a common surgical emergency in India. Despite advancements in antimicrobials and supportive care, mortality associated with diffuse suppurative peritonitis is high. The Mannheim Peritonitis Index is a specific score, which has a good accuracy and has ease of handling of clinical parameters. It allows for easy prediction of the prognosis in patients with peritonitis.

Methodology:

The study is done in 50 patients with peritonitis due to hollow viscous perforation who presented to PSG Hospitals, Coimbatore. MPI score was calculated for each patient and the post operative course followed up.

Result:

In our study, appendicular perforation was found to be the most common cause of perforation peritonitis accounting for 26 percent of the cases. This is followed by perforation of peptic ulcer (20 %) and traumatic perforations (16%). 64 % of patients with peritonitis had MPI score of less than 21. These patients had a morbidity rate of 34.37. Thirty percent had

MPI score within 21 to 29. These patients had a morbidity rate of eighty percent. There were three patients who had MPI score of above 29. Two of these patients died and the remaining one had post op morbidity. There was no mortality in the other two groups. The association of increasing MPI score with mortality and morbidity is found to be significant. The p value is <0.001. Duration of peritonitis, age > 50 years, presence of organ failure, site of perforation, extent of peritonitis and the nature of peritoneal exudate were found to be positively associated with the prognosis.

Conclusion:

The most common causes of perforation peritonitis were found to appendicitis and peptic ulcer disease. It has been found that the Mannheim peritonitis index has been a good predictor of mortality as well morbidity in patients with peritonitis.

Key Words :

Mannheim Peritonitis index, MPI scores, Perforation Peritonitis, Peritonitis, Prognosis in peritonitis, Spectrum of Peritonitis.

INTRODUCTION

INTRODUCTION

Perforation peritonitis has been found to be a common surgical emergency in India. The causes of perforation in India have been found to be quite different from that in western countries.[1] But there is a lack of data about the etiology and the morbidity and mortality patters in cases of perforation peritonitis from India. [2]

It has been found that the prognosis of patients with peritonitis and intra abdominal infections is poor. This is especially so when multi organ failure sets in.

Despite advancements in antimicrobials and supportive care, mortality associated with diffuse suppurative peritonitis is high. Accurate diagnosis and management of suppurative peritonitis is a challenge. Complex surgical interventions, multifaceted treatment aspects and difficulties of ICU support make evaluation of new therapeutic advances very difficult in this field.

In these situations scoring systems which provide accurate assessment of the patient's conditions at a specific point in the disease simplifies the understanding of these problems. These scoring systems serve as a prognostic marker and help us evaluate our line of management.

In past many scoring systems have been devised to help assess the prognosis in patients who are critical ill. The evaluation of patients with peritonitis proves to tougher because of the varied etiologies, treatment modalities.

Of the many scoring systems the Mannheim Peritonitis Index which was developed by Wacha[3] and Linder in 1983 was found to be one of the simplest scoring systems that easily allows the surgeon to predict the outcome in patients with peritonitis. The MPI score was based on the analysis of retrospective data from 1253 patients with peritonitis. A total of 20 possible risk factors were considered. Of these only 8 proved to be of prognostic relevance and were entered into the Mannheim Peritonitis Index.

The Mannheim Peritonitis Index is a specific score, which has a good accuracy and has ease of handling of clinical parameters. It allows for easy prediction of the prognosis in patients with peritonitis.[4]Also collection of retrospective data is valid and possible, as the Mannheim Peritonitis Index only requires data that are routinely found in surgical registers.[5]

Understanding the patho physiology of peritonitis, the concept of sepsis and multiorgan failure has furthered the management of peritonitis. Current trends focus on early identification of the potential candidates who are prone to have a indolent course and to start aggressive therapy in these subset of patients. In patients who have progressed to multi-organ failure conservative treatment and newer modalities of treatment such as immuno modulation and programmed relaparotomy are being tried.

AIM OF THE STUDY

AIM OF THE STUDY

- To assess the effectiveness of the Mannheim peritonitis index in predicting the outcome of patients with peritonitis
- To assess the significance of each risk factor of the Mannheim index in predicting the prognosis
- To assess the morbidity and mortality rates in patients with peritonitis
- Evaluate various conditions leading on to peritonitis

REVIEW OF LITERATURE

REVIEW OF LITERATURE

HISTORY

Physicians in the past dreaded abdominal complications. Despite peritonitis being extremely common, reports of successful surgical interventions were rare before the past century. Medicine's comprehension of the pathophysiology of peritontis is still evolving. Despite this, the mortality rates for secondary peritonitis have fallen in the last century from almost 100% to less than10%.

Earliest references to peritoneum can be found in Edwin Smith Papyrus around 1700 years ago which is supposed to have been written around the time of Imhotep (the Egyptian patron god of medicine).

Since the time of recorded medical history, humans have been confronted with the various presentations of peritonitis. Accounts from early societies have little doubt that our predecessors recognized the value of therapeutic drainage. In a German translation of the writings of Hippocrates is portrayed the first through description of a patient with peritonitis. He described septic shock as *"A protrusive nose, hollowed eyes, sunken temple, cold drawn in ears with outturned lobes, the forehead's skin is rough and tense like a piece of parchment and the whole face greenish or black"*.

In the second century A.D. Galen physician to the Roman citizens, gladiators and emperors is said to have performed many surgeries including suturing of

lacerated bowel loops. He wrote about appearance of suppuration in post-operative period. In fact, Galen believed that such suppuration was important for proper and faster wound healing and should not be drained (laudable pus). Galen's writings were revered and restrained the development of medicine and physiology for almost 1500 years.

From the fall of the Roman Empire to the beginning of the 16Th century, medicine can be characterized as magical with religious overtones. The fate of surgery was sealed for many centuries with the Pope Innocent III religious decree of 1215 which was known as *"Eccelsia Abhorret de Sanguine"*, translated as *"The Church Abhors bloodshed"*. It was only at the birth of renaissance that the mysteries of the abdominal cavity began to unravel. This is attributed to the beautiful drawings of the Michelangelo, Leonardo da Vinci and Vesalius.

Peritonitis due to acute peptic ulcer with perforation was first described by Littre in 1670. Hertein, in 1767, reported curing biliary peritonitis in dogs by using abdominal irrigation.

The three major developments that fostered an understanding of the disease process of peritonitis included the founding of experimental physiology by Francois Magendie and Claude Bernard, an understanding of cellular pathology as written by Rudolph Virchow, and the germ theory by Pasteur and Koch.

George Wegner reported first in 1879, a series of experiments attempting to explain the normal physiology of the peritoneum. The modern era of understanding the peritoneum was begun by John B. Murphy. In 1908, he wrote *"There are no stomata or stigmata in the peritoneum. The endothelial lining is everywhere and continuous"*. [6] Of course, we know it is not completely right as of today. Herbert E Durham[7] analyzed peritoneal fluid and proposed a time line of cellular events, which were divided into 5 stages – (1) the stage preceding leukopenia, (2) the leukopenic stage, (3) the microxyphil stage, (4) the macrophage stage and (4) recovery to normal cellularity.

The experiments of Meleney [8] in 1926 showed the existence of bacterial synergism. They showed that combinations of aerobic and anaerobic bacteria produced more sepsis than individual strains.

Review of Current Literature

Few of the early attempts to define the severity of surgical infection and chances of death came from the observation that patients who died after surgical infection often followed a clinical course that was characterized by sequential failure of vital organs.This was termed**"multiple organ failure syndrome".**

Fry and associates [9] demonstrated in 1980 that death after major operations or extensive trauma was usually as a result of infection and the risk increased as the

number of failed organs increased i.e. the mortality rate without organ failure was 3%, increasing to 30% in 1 Organ failure and 100% in 4 organ failure.

In 1982 Knaus and others proposed a scoring system to be used for classifying patients admitted to intensive care. They devised a 2 part scale. It was called APS-34 and examines the abnormality among 34 possible physiological assessments, which were acquired during the first day of admission. The second part of the score was a chronic health evaluation (CH). This determines the patient's pre-admission health by examining the medical history for details concerning functional status, productivity and medical attention in the preceding 6 month before admission. The combination is called APACHE. This system was not specific to intra-abdominal infection. It was later modified using only 12 values as the APACHE II.

Another approach to grade the severity of sepsis was published by Elebute and Stoner in1983[10]. They divided the clinical features of the sepsis into 4 classes to which were ascribed subjective degrees of severity based on an analogue scale. The attributes were as follows: local effects of tissue infection, degree of temperature elevation, secondary effects of sepsis and lab values.

Pine and associates[11] (1983) confirmed the findings of Elebute and Stoner. In addition, they also looked at a number of other risk factors influencing the development of organ failures on death and concluded that clinical shock at any time, malnutrition, alcoholism and age were important predictive factors. The

papers by Pine and Knaus and their colleagues were the first to give the clear definition of **"organ failure".**

Knaus and Coworkers[12] (1985) seconded these observations in a report covering 5,677 ICU admissions and 2719 patients who developed organ failure.

Teichmann and associates[13] (1986) in a report concerning scheduled reoperation for diffuse peritonitis, referred to Peritonitis Index Altermheir (PIA). This used age, extent of infection, malignancy, cardiovascular risks and leukopenia to grade patients.

Wacha and Coworkers[3] in 1987 developed a separate index for peritonitis called the Mannheim Peritonitis Index (MPI) by incorporating information regarding age, gender, organ failure, cancer, duration of the peritonitis, involvement of colon, extent of peritoneal spread and the character of peritoneal fluid to define risk. Scores range from 0 to 46.

In 1988, V. Kohli[14] and others evaluated the prognostic factors in 50 cases of perforated peptic ulcer causing peritonitis. They found that General Health, presence of concurrent illness, arterial hypotension at the time of admission, delay in surgery and the severity of peritoneal contaminations as some of the factors that contributed to the post-operative morbidity and mortality.

In 1990, Verma and others[15] from PGI, Chandigarh, compared the prognostic factors in peritonitis secondary to trauma. They found that pre-operative shock,

multiple hollow visceral injury, septicemia, and location of injury (colon and duodenum were significant prognostic factors and with high mortality) played a major role in the prognosis.

In 1992, Bartel and others did a study of the use of programmed relaparotomy in diffuse peritonitis. They concluded that the eradication of source of infection during first laparotomy, serum creatinine, patients age and pre-existing hepatic disease influenced outcome.

Khosrovanin in 1994 identified 3 important prognostic factors causing high mortality they were - age over 70 years, admission delay of > 24 hours and pre-operative hemodynamic shock. He recommended the suture of perforation and vagotomy in absence of risk factors.

In 1994, Kriwanek S. conducted a study for the prognostic factors in colonic perforation. It concluded that age over 65 years and MPI proved to be the only risk factors of prognostic significance.

In 1994, Scoanes[16] and others did a study of diverse effects of delayed treatment for perforated peptic ulcer. They concluded that delaying treatment for > 12 hrs increased the mortality especially in elderly patients confirming the findings of MPI.

In 1996, a multivariate analysis on 604 patients with intra-abdominal infection was

done to compare different scoring systems like Apache-II, SS of Elebute and Stoner and MPI. Results showed dominance of host-related factors over the type and source of infections on the prognosis of patients. Apache-II and MPI scores were able to predict the outcome correctly.

SURGICAL ANATOMY OF PERITONEUM AND PERITONEAL CAVITY

Embryology of peritoneal cavity:

The peritoneal cavity is formed from the intraembryonic coelom. Tne intraembryonic coelom is horseshoe shaped. It is present caudal to the septum transverses. The intraembryonic coelom has two limbs which are separate at first. Due to lateral folding of the embryonic disc, the two parts fuse to form one cavity. Initially the mesentery of the primitive gut is attached to the posterior abdominal wall in the midline. The attachment gets complicated due to the changes arising from the rotation of gut, as some parts of the gut then become retroperitoneal. [17] The splanchnic mesoderm enclosing the primitive gut forms the lining of the peritoneal cavity. Mesenteries and the ligaments of the visceral organs are formed by double layers of the peritoneum.[18] The peritoneal cavity is thus separated by folds of peritoneum into a number of pockets.

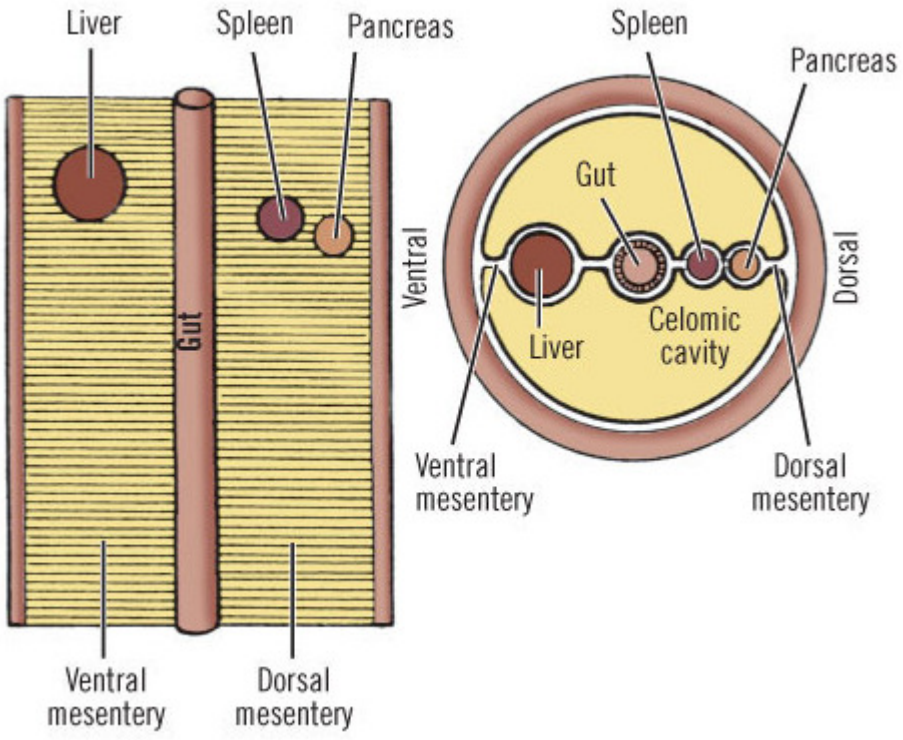


Figure 1 Relationship of various organs with embryonic ventral and dorsal mesenteries

Parietal peritoneum:

The parietal peritoneum is the layer of peritoneum that coats the inner surface of the walls of the abdomen and pelvis, and the inferior surface of the diaphragm. It has somatic innervations and is hence pain sensitive. It can easily be separated from the abdominal wall as it is loosely attached by extraperitoneal connective tissue.

Visceral peritoneum:

Visceral peritoneum is the layer of peritoneum coating the abdominal viscera. It derives its blood and nerve supply from the viscera that it covers. Hence the

visceral peritoneum is pain insensitive as it has got autonomic innervations. It is firmly adherent to the viscera it covers and cannot be separated from it.[19] Histologically, peritoneum has two layers. An outer layer which is composed of fibrous tissue, thus providing strength to the peritoneal membrane. The inner layer is composed of mesothelial cells, which secrete the serous peritoneal fluid. This fluid acts to lubricate the peritoneal cavity.

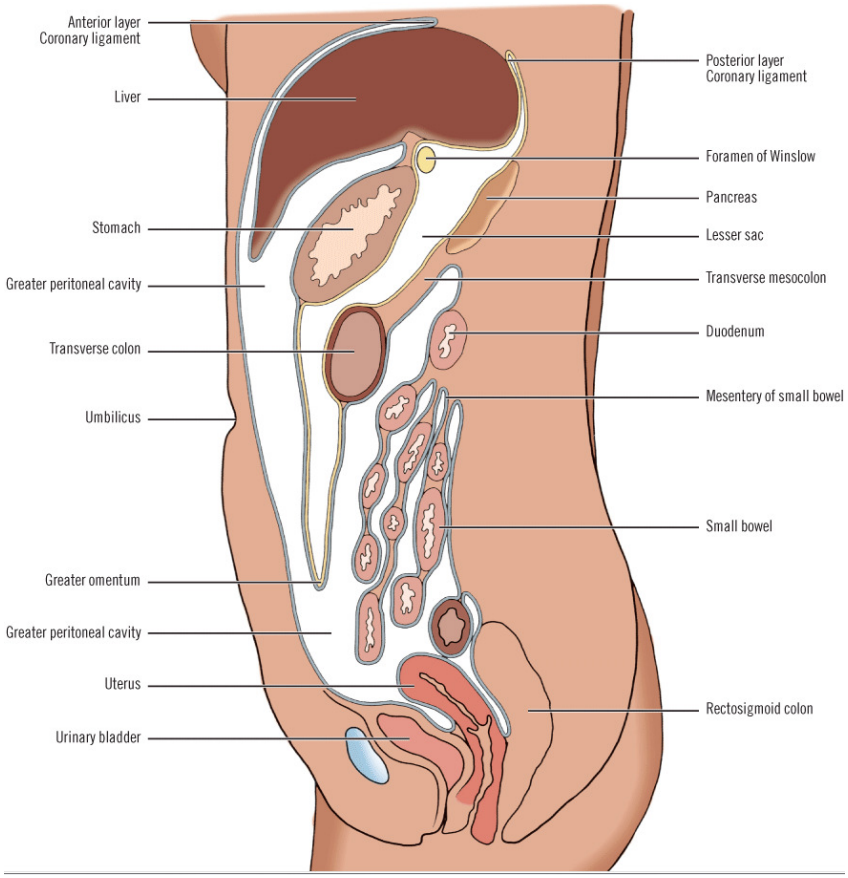


Figure 2 Vertical disposition of the peritoneum

The peritoneal cavity has a surface area nearly the same as skin. It is about two square metre in adults. The peritoneal cavity is a closed structure in males. The free ends of the fallopian tubes open directly into the peritoneal cavity in females. The peritoneal cavity is divided into pelvic and abdominal portions. The abdominal portion is divided into supracolic and infracolic compartment by transverse colon and mesocolon. The infra colic compartment is divided into right and left by mesentery. The Right infracolic and left infracolic is divided into external and internal paracolicgutters by ascending and descending colon respectively. Supracolic compartment is below the diaphragm and above transverse colon and mesocolon. The liver, gallbladder, stomach, first part of the duodenum and spleen lie in this space. The liver and its ligaments break this space into important sub phrenic spaces.

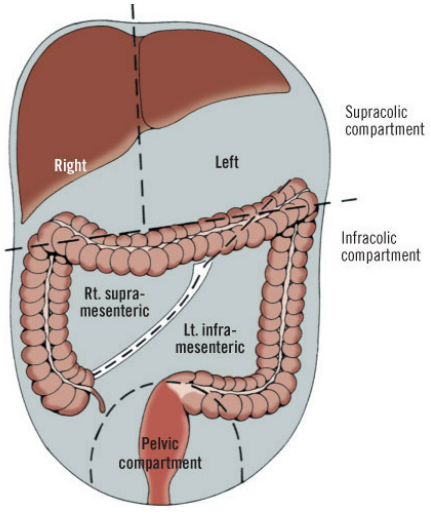


Figure 3 Compartments of abdominal cavity

Pelvic cavity:

This is a funnel shaped posteroinferior extension of the abdominal cavity proper and the most dependent part of the peritoneal cavity. Bounded anteriorly by the hip bones and the obturator internii muscles, posterosuperiorly by the sacrum, coccyx, piriformis and coccygeii, inferiorly by the levator ani and the urogenital diaphragm it contains the urinary bladder, the terminal parts of ureters, the sigmoid colon, the rectum, some ileal coils, the internal genitalia, vessels, lymph nodes and nerves.

Peritoneal microstructure:

Peritoneum consists of a single layer of mesothelial cells which overlies a layer of loose connective tissue. The mesothelial cells are polyhedral in appearance and are flattened. They have microvilli and cilia which serves to increases the surface area for absorption and also aids in the circulation of peritoneal fluid in an upward direction.[20]This mesothelial lining acts as a dialyzing membrane. The mesothelial cells also contain many vesicles. These vesicles aid in fluid transport by the process of endocytosis. The basement membrance of loose collagen fibres lie beneath the mesothelial cells. The basement membrane in turn lies over a complex connective tissue layer. The connective tissue layer contains collagen and other connective tissue proteins, elastic fibres, fibroblasts, inflammatory cells, adipose cells and a network of lymphatic vessels and capillaries.[21 22]

Peritoneal fluid

Usually the peritoneal cavity contains less than 50 ml of fluid. The peritoneal fluid is secreted by the visceral peritoneum. The peritoneal fluid is pale yellow in color. It is absorbed by the parietal peritoneum and diaphragmatic lymphatics. The specific gravity of the peritoneal fluid is low and it has a low protein content and less than 3000 cells per cubic millimeter. Water, electrolytes and other solutes in the peritoneal fluid are derived from the interstitial fluid and from the plasma. The cellular components include peritoneal macrophages, fibroblasts, mast cells, desquamated mesothelial cells, lymphocytes and some other leucocytes. Neutrophils are found to be absent in the peritoneal fluid. In various pathologies afflicting the peritoneum, the cellular content is found to vary in number and type. Hence the analysis of the cellular content is of diagnostic significance. The absorption of the peritoneal fluid occurs through both capillaries (through which solute content is absorbed) and lymphatics (through which suspended particulate matter is absorbed)[23]

PHYSIOLOGY OF PERITONEUM

The peritoneum serves to maintain the surface integrity of intraabdominal organs and provides a smooth and lubricated surface for the intestine to move freely.[23]

Lymphatic drainage

The parietal lymphatics drain almost 70% of the contents of the peritoneal fluid. The remainder 30% including suspended particulate matter and microorganisms are drained through the diaphragmatic lymphatics. There is uniqueness in the anatomical and physiological arrangement of the mesothelial cells which overlie the diaphragm. The cells are interrupted by intercellular gaps or the stomata.[24] These stomata are present only over the muscular section of the diaphragm. They act as gateways to a well developed plexus of diaphragmatic lymphatics. These lymphatic channels are called lacunae. They contain valves that serve to prevent reflux. [25] The lymph passing through the diaphragmatic lymphatics circulate to the substernal lymph nodes and then ultimately drain into the thoracic duct.

The stomata have an average size of 4 —12μm.

The factors which influence the absorption of fluid through the diaphragmatic lymphatics include

(a) action in the mesothelial cellular process, which when decreased causes increase in stomata size

(b) inhalation , during which there is diaphragmatic contraction and descent which causes contraction of stomata. Also there develops negative intrathoracic pressure that aids emptying of the lacunae into thoracic lymph channels.

(c) exhalation, during which there is diaphragmatic relaxation and opening of stomata

(d) increase in intra abdominal pressure which causes the stomata to remain patent

(e) inflammation of the peritoneum which causes increase in stomata size.[23]

The negative pressure area in the subphrenic space due to diaphragmatic motion accounts for the cephaland movement of the peritoneal fluid. These account for the higher incidence of subphrenic abscess in the bedridden patient with peritonitis. The transit time for bacteria from the peritoneal cavity to the blood steam via this route is estimated at six minutes and is retarded by the head up position, as well as diaphragmatic paralysis. Prior to the use of operative therapy for peritonitis, a study reported a decrease in the rate of mortality due to peritonitis when the patient is nursed in the semi-upright posture.

The peritoneal surface other than that over the diaphragm acts as a passive semipermeable membrane. It allows for bidirectional free exchange of fluid and solutes. The peritoneum over the diaphragm in addition can also allow for the passage of particulate matter due to presence of stomata. The stomata are four to twelve micrometers in diameter, but are capable of expanding to accommodate

particles upto even 23 micrometers. Faster absorption is noticed with smaller particles though.

Defense Mechanisms:

The defense mechanisms by which the peritoneum deals with the invading pathogens are

i)Translymphatic absorption by which bacteria are directly removed

ii) destruction of bacteria through activation of complement system and by phagocytosis.

iii)preventing systemic infection by sequestering the infection through fibrin trapping, omental containtment and ileus.[27]

Peritoneal Response to Injury:

There is loss of mesothelial cells at the site of peritoneal injury. It has been found that the time taken for a peritoneal defect to heal is the same no matter what the size of the defect is, the time usually being 3-5 days.[28] After peritoneal injury, it has been found that the wound gets covered by connective tissue cells by the third day. By day 5, the cells resemble adjacent normal mesothelium. The mesothelial regeneration is complete by day 8. The cells responsible for the mesothelial regeneration are yet to be identified. Possibilities are that the mesothelium regenerates from a. Submesothelial stem cells b. Free floating mesothelial cells

c.Mesothelial cells attached to the wound margins d.Monocytes and macrophages differentiating into the mesothelial cells.[29]

The visceral and parietal peritoneum appear to have similar mechanisms of wound healing.[30]Normally peritoneum heals without adhesion formation. Adhesions occur as consequence of factors other than just peritoneal injury. The most important factor is local tissue hypoxia and ischemia. Injury to the submesothelial surface and intraabdominal infection are other factors that favour the formation of adhesions.[29]

The peritoneum lyses fibin deposits by an enzyme system. Injury to the peritoneal surface halts this activity. Return of enzymatic activity takes a couple of days.

In a majority of patients who undergo abdominal surgery, intra peritoneal adhesions take place. Adhesions also occur due to other causes which result in peritoneal injury.[31] Intra peritoneal adhesions are the most common cause for the development of intestinal obstruction in developed countries.[32 33]

In patients who have inflammation of the peritoneum, ischaemia of the peritoneum and other conditions predisposing to adhesions, it has been noticed that there is a decreased plasminogen activating activity(PAA). This supports the hypothesis that decreased PAA leads to formation of fibrinous adhesions.[31]

In finality, the resolution of infection depends upon the critical interaction between the invading pathogen and the immune system within a microenvironment that is fibrin laden.[34]

Classification of peritonitis

Primary peritonitis

a. Spontaneous peritonitis in children

b. Spontaneous peritonitis in adults

c. Peritonitis in patients with continuous ambulatory peritoneal dialysis.

d. Tuberculous peritonitis.

e. Other forms.

Secondary peritonitis:

a) Acute perforation peritonitis:

1. Gastrointestinal tract perforation
2. Bowel wall necrosis
3. Pelvic peritonitis
4. Other forms.

b) Postoperative peritonitis

1. Anastomotic leak
2. Leak of a simple suture
3. Blind loop leak
4. Other iatrogenic leaks

c) Post traumatic peritonitis

1. Peritonitis after blunt abdominal trauma
2. Peritonitis after penetrating abdominal trauma

Tertiary peritonitis

Primary peritonitis[35]

It refers to peritonitis that occurs in the absence of GI perforation. It occurs by haematogenous spread , though, occasionally the infection can occur by transluminal spread or direct invasion. It is commonly associated with cirrhosis and advanced liver disease. It is also seen to occur in patients with nephrotic syndrome, systemic lupus erythematosis or after splenectomy during childhood.

Secondary peritonitis

Perforative peritonitis

Perforation of the gastrointestinal tract leads to secondary peritonitis. Perforations of the stomach and duodenum have acute and sudden presentations. Initially the peritonitis is sterile but eventually infection sets in.

IN case perforations involving the small intestine the presentation is either that of bowel obstruction followed by peritonitis or necrosis of the bowel wall with perforation. Cases of mesenteric ischaemia are usually diagnosed late and have a high mortality rate. Perforation in cases of typhoid usually occurs in the third week of infection. The Peyer's patches undergo inflammation, hypertrophy and subsequently haemorrhage and perforate. In cases of appendicitis, peritonitis is usually localized and generalised peritonitis is rare.

Perforation involving the colon accounts for 22 % of peritonitis cases.

Inflammatory pathology such as diverticulitis, colitis account for 50%, and rest are due to luminal or external obstruction. The commonest sites of perforation are the ascending colon and the caecum.

Perforation resulting from amoebic infection can occur as consequence of ruptured liver abscess or perforation of colon.

Peritonitis due to pancreatitis is usually managed conservatively. In the setting of infected necrosis or severe necrosis not responding to conservative management, surgical debridement is necessary.

Post operative peritonitis is the result of anastomotic site leak. It is usually diagnosed between 5^{th} and 7^{th} post operative day. It can be managed by drainage and controlled fistula formation. Some times reexploration with resection and reanastomosis or stoma formation may be necessary. Staged abdominal repair may be required.[36]

Posttraumatic peritonitis

Blunt trauma to the abdomen can cause hollow viscus perforation or mesenteric rupture leading on to loss of intestinal blood supply and subsequent bowel ischaemia. Diagnosis of a bowel injury may be delayed in the setting of a poly trauma especially in patients with concomitant head injury. Likewise penetrating trauma can cause perforation or mesenteric injury.

BACTERIOLOGY

Bacteriology of peritonitis

Perforation of the GI tract is the commonest cause of contamination of the peritoneal cavity. Contamination can occur from other sources such as penetrating trauma or secondary contamination from infected viscera or septicaemia.[37]

The type of pathogen depends on the site of perforation. Perforations involving the stomach and duodenum usually cause sterile peritonitis due to the presence of gastric acid. In case of perforation of gastric ulcer, which is associated with hypoacidity, infection is by gram positive anaerobes from the oral cavity, candida species and gram negative bacilli.

The terminal ileum and the colon are sites of high bacterial load and contain more than 400 species of bacteria. Anaerobic bacteria are more in number as compared to aerobic bacteria, the ratio being 100:1. Among the aerobic pathogens, the most common is Escherichia coli. Enterococcus is the principle gram negative facultative bacteria. Klebsiella, Proteus and Pseudomonas are other gram negative facultative organisms of significance. The most important anaerobic pathogen involved in intra abdominal infection is Bacteroides fragilis.[38] Other anaerobic pathogens of significance are Clostridia, Peptostreptococci, Peptococci, Fusobacteria and Veilonella.[37]

Weinsten and associates conducted a series of studies where in colonic peritonitis was studies in rats by introducing pooled rat caecal contents mixed with barium into the peritoneal cavity. Introduction of the innoculum was followed by acute generalized peritonitis with a mortality rate of 40%. During this period of acute peritonitis, 95% of the animals had bacteremia due to E coli. Those animals which survived developed a second more benign stage during which there developed multiple intra abdominal abscesses. The original innoculum, on culture, grew more than 27 species of bacteria. Culture from the peritoneal cavity grew primarily E.coli, B.fragilis and Enterococcus. It was found that during the acute peritonitis phase, E.coli and Enterococcus were predominant pathogens and B. fragilis was the dominant pathogen during the abscess phase. Synergy among the bacteria is important since the intra abdominal infections almost always harbor polymicrobial flora. Experimental studies have revealed that animals were able to tolerate infection by single organism, but a combination of various pathogens lead to mortality. [23]

It is clear that anaerobes are the predominant pathogens in the development of intraabdominal abscesses. But their role in causing sepsis is unknown. B. fragilis is innocuous when it is the only pathogen. But in the presence of other virulent organisms it can serve to increase their pathogenicity, especially that of E.coli. it

has been found that anaerobes such as B.fragilis drastically inhibit the function of neutrophils by secreting large amount of succinic acid.[39]

In an oxygen rich environment, the growth of bacteroides and other anaerobes are hampered. Enterococci , E coli and other facultative organisms by virtue of their rapid aerobic growth lower the local redox potential and thereby facilitate the growth of anaerobes. [23]

Table 1 : Bacteria commonly encountered in peritonitis

Facultative anaerobe and Gram-negative aerobic	Obligate Anaerobes	Facultative anaerobic gram-positive aerobic
Escherichia coli	Bacteriodes fragilis	Enterococci
Klebsiella species	Bacteriodes species	Staphylococcus
Proteus species	Fusobacterium species	Streptococcus
Enterobacter species	Clostridium species	
Morganella morganii	Peptococcus species	
Aerobic gram-negative bacilli	Peptostreptococcus species	
Pseudomonas aeruginosa	Lactobacillus species	

Paths to peritoneal infection:

The peritoneal cavity is infected through the following routes

• perforation of the GI tract

• Exogenous contamination via drains, open surgery, trauma.

• Transmural translocation of bacteria for eg. Inflammatory bowel disease, bowel ischaemia, appendicitis.

• infections from the female genital tract

• haematogenous spread i.e. Septicaemia.

PATHOGENESIS

Response of peritoneal cavity to infection:

Soon after infection of the peritoneal cavity, a local response in mounted. Initially there is hyperaemia, followed by influx of fluid and inflammatory cells and fibroblasts. Peritonitis is classically stimulated by Gram negative bacteria associated endotoxins .Other factors causing mesothelial or vascular endothelial cell injury and bacterial exotoxins are also capable on initiating peritonitis.[40]

The peritoneal cavity responds to inflammatory insult immediately by numerous non specific inflammatory reactions.

The peritoneum usually allows for bidirectional flow of fluid. With the onset of the inflammatory process flow changes to an unidirectional one. Fluid of volume 10

litres or more can accumulate, depending upon the extent of peritoneal involvement.[41]

The surface area of the peritoneum is about 1.8 metre square. An 1 mm Increase in the thickness can cause accumulation of about 18 litres of fluid. Transudative fluid is accumulated first following inflammation. Later an exudative fluid rich in inflammatory mediators accumulate.[42]

This exudative fluid proves to be a double edged sword. It helps deliver the essential humoral factors of inflammation. But the downside is that the massive third space loss caused hypovolumeia. There is also dilution of opsonin, impairment of bacterial phagocytosis and impedence of neutrophil mobility.[43]

The diaphragmatic lymphatics, the peritoneal macrophages and the influx of neutrophils are the three major mechanisms by which the peritoneal cavity tries to clear itself of the offending pathogen. The diaphragmatic stomata enlarge after an inflammatory stimulus due to retraction of the mesothelial cell processes. This causes an increase in the patency of the stomata which persists for as long as three days. Increased stomata patency in combination with the clearance function of diaphragm causes rapid clearance of bacteria from the peritoneal cavity into the blood stream. Thus begin the systemic effects of sepsis which serves to further pump bacteria away from the peritoneal cavity due to the development of tachycardia. [44]

The primary mechanisms for bacterial clearance are the diaphragmatic lymphatics and the macrophages. The neutrophil influx acts as second tier defence, which acts against those remaining pathogens.[23]

The neutrophil influx becomes significant by 4-6 hours and by eight hours it reaches a peak.[45]The influx of neutrophils is enhanced by activated resident macrophages.[46]Local fibrinolytic activity is suppressed following mesothelial injury. The injured cells release thromboplastin which enhance fibrin deposition through the intrinsic pathway. The stimulated peritoneal macrophages also play a role in increasing the surface procoagulant activity. The purpose of fibrin deposition is to attempt to contain the infection by trapping the bacteria. It also causes adhesions between bowel loops and parietal peritoneum, thereby preventing bacterial contamination spread. [47]That the fibrin encasement of the bacteria also isolates the bacteria from neutrophils is the problem.[48] Mesothelial regeneration causes lysis of early fibrinous adhesions. But in cases of severe peritoneal injury there is persistent inflammation which impairs fibrinolysis. There occurs invasion by fibroblasts and neovascularisation develops with the purpose of increasing blood supply to the site of ischaemia. This leads to fibrinous adhesion formation within a period of about ten days from the onset of injury. With passage of time though, these adhesions may disappear.

The development of abscess within the adherent mass occurs as a consequence of proteolysis by leucocytes and lysis by bacterial exoenzymes.

Bowel response

The primary bowel response to the peritoneal inflammation is transient hypermotility. Subsequently motility is depressed which is followed by adynamic ileus. The purpose of decreased bowel motility is to prevent the spread of infective fluid. Due to decreased motility there is sequestration of fluid and air intraluminally causing bowel distension.[50]

Abdominal wall reaction

There is reflex contraction of the abdominal muscles in response to inflammation of the parietal peritoneum. This splinting of the abdominal wall serves to limit the spread of infective fluid. Also, since the parietal peritoneum is pain sensitive, the patient contracts the abdominal muscles voluntarily.

Factors influencing the local response

Bacterial virulence

Several factors influence the virulence of the contaminating bacteria. Phagocytosis is inhibited by capsulated organisms.[51] The anaerobes which account a great proportion of the colonic flora rarely are implicated in intra abdominal infection. The most common pathogen amongst the anaerobes is Bacteroides fragilis which

accounts for only one percent of the colonic flora.[50]Some bacteria have an innate capacity to cause peritonitis, such as E coli and Enterococcus. Organisms such as B. fragilis, on the other hand, have a tendency towards formation of intraperitoneal abscess formation by virtue of their capacity to adhere to the peritoneum. There is proportionate relationship between size of the bacterial innoculum and the ability to produce infection and subsequently the disease severity. The ability of B.fragilis and enterobacteriaceae to adhere firmly to the mesothelial cells renders them resistant to mechanical removal by lavage. As mentioned earlier, bacterial syngerism in the setting of poly microbial infection is also an important factor.

Gastric juice

Gastric juice contains hydrochloric acid, mucin and other digestive enzymes, all of which are potent irritants of the peritoneal cavity. The acidic nature of gastric secretions causes death of bacteria thereby rendering them sterile. But in the setting malignant ulcers with hypo or achlorhydria, suppurative peritonitis may ensure in the event of a gastric perforation.

Pancreatic juice

Pancreatic juice causes peritoneal irritation. The peritonitis in the setting of pancreatits is sterile to begin with. There may be bacterial infection following bacterial translocation from within the colon. Trypsinogen which is found in

pancreatic juice, is converted to active form by bacterial action. This causes tissue digestion and subsequently increased bacterial invasion.

Bile

Bile is found to have less irritant action on the peritoneum. But it causes reduction in the surface tension and enhances spread of bacterial infection. Bile salts also function as culture medium for bacteria such as Enterococcus fecalis and are also toxic to neutrophils.[49]

Urine

Urine though sterile is highly irritant to peritoneum. Also the resorption of urea and acidic metabolites causes acidosis and uremia.[52]

Hemoglobin

The haemoglobin ferrous ion that is released following red cell lysis acts as mild osmolar irritant. It also impairs the phagocytic activity and enhances the virulence of E coli by acting as a strong culture medium

Products of inflammation

Fibrin inhibits phagocytosis, blocks diaphragmatic lymphatics (with platelets) and leads on to premature degranulation of neutrophils. The formation of large volume of intra peritoneal fluid due to peritoneal inflammation causes the dilution of opsonins and bacterial dispersion. It renders the phagocytic activity of macrophages as ineffective since they require a surface to act.[43]

Extent and Duration of Contamination

Acute contamination of the peritoneal cavity by large volume of contaminant as which occurs during rupture of caecum in cases of carcinoma causes rapid dispersion of bacteria into the peritoneal cavity. Subsequently the nature of peritonitis is severe in such cases. Slow oozing of GI contents from small perforations in the proximal GI tract casues less severe form of peritonitis

Site of Perforation

In cases of perforation involving the stomach and the small bowel till the proximal ileum, only small volume of infective material enters the peritoneal cavity. But the contaminant is fluid in nature which makes localization difficult.

In cases of perforation of distal ileum and caecum, the contaminant released into the peritoneal is fluid in nature, of large volume, and has high bacterial load. Also there are residual active enzymes which act as adjuvants. Therefore distal ileal and caecal perforations are the most dangerous and are associated with greater morbidity.

Factors promoting diffusion :

If perforation occurs before the protective mechanisms have been initiated, there is a rapid release of intestinal contents into the peritoneal cavity. This contaminant spreads over a large area of the peritoneum almost instantaneously

Ingestion of food or water by causing peristalsis hampers localization of infection. Likewise violent peristalsis caused by administration of enema or a purgative also hinders localization.

Secondary Responses in Peritonitis

Cardiac response

There is fall in circulatory volume due to sequestration of fluid in the peritoneal cavity. This leads to fall in cardiac output resulting in hypotension and poor oxygenation of tissues. This leads to metabolic acidosis which further depresses the cardiac function.[53]

Renal changes

Changes in the renal system are due to hypovolumia. Due to fall in cardiac output, there is increased secretion of the anti diuretic hormone and aldosterone. These factors lead to decreased renal perfusion. There is a fall in the glomerular filtration rate which leads on to renal insufficiency. Ultimately this causes development of metabolic acidosis.[54]

Respiratory changes

There is a fall in the tidal volume due to distension of the abdomen caused by ileus and due to restriction of diaphragmatic movements caused by pain. These factors

predispose to the development of atelectasis. Atelectasis in turn causes ventilation – perfusion mismatch and the partial pressure of oxygen in blood falls.[55]

Hormonal and metabolic changes

There is secretion of large amount of epinephrine and nor epinephrine into the blood. This causes vasoconstriction, tachycardia and sweating. The first three days following onset of peritonitis, there is also increased secretion of the adrenocortical hormones. Antidiuretic hormone and aldosterone are secreted in increased quantity which causes reduction of urine volume and conservation of sodium and water. The retention of water may be more than that of sodium leading on to dilutional hyponatremia. The metabolic rate in individuals with peritonitis is increased. This results in increased oxygen demand also. But due to presence of hypovolumia, reduced cardiac output and decreased respiratory efforts, tissue perfusion is hampered. Thus a supply – demand disparity is established. Patients develop latic acidosis. The catabolism of protein is also increased. There is a progressive fall in the serum albumin level due to accumulation of albumin in the peritoneal cavity.[56]

PROGNOSIS OF PERITONITIS

1. Age:

With increasing age there is impairment of the host defence processes. There is a decreased delivery of phagocytes to sites of contamination by the bacteria.[57]

The following changes that occur during aging are significant in relation to the spread of infection

i. The thymus gland reduces in size leading to fall in the levels of the mature T-Lymphocytes.

ii. the chemotactic and phagocytic activity of polymorphonuclear leukocytes are reduced

Bohnen and Boulangere conducted a study on 176 patients with generalised peritonitis which showed that patient over 50 years had 45% mortality rate as compared to patients lesser than 50 years who had only 17% mortality rate.

Age was found to not have a bearing in cases of post operative peritonitis.[58]

Kaltarenzos and associates made a study which analysed 42 patients of severe peritonitis. It was found that patients above 65 years of age had a mortality rate of 33% .[59]

2. Source of infection

In cases of generalized peritonitis, the source of contamination was found to be an important prognostic factor. In perforation of a duodenal ulcer or gastric ulcer, the mortality rate was found to range between 9 to 40 per cent.[60]

A study was conducted among 44 patients with generalized peritonitis and the following observations were noted :

Colonic perforations had a mortality rate of 54%, small bowel perforation a 21% mortality rate and perforated gastric & duodenal ulcer a 12.5% mortality rate.

3. Duration of preoperative illness

The time duration of peritonitis before surgical intervention has a remarkable effect on the outcome of the patient. This is mainly due to the increased incidence of preop septic shock in patients who have a delayed medical intervention.[61]

Delayed intervention causes overgrowth of the gram negative bacteria and facilitates synergestic poly microbial growth.A study was made in 44 patients with generalized peritonitis. The study concluded that that there was more than a two fold increase in mortality between patients who underwent surgery within 24 hours of illness and those who underwent surgery later than 24 hours after the onset of symptoms.

4. Associated chronic diseases

Patients who have chronic diseases such as diabetes, have a poorer outcome. Diabetics have an immune suppressed state which arise from the decreased ability of the polymorphonuclear leucocytes to destroy the bacteria engulfed by them.[62] Also the activity of leukocytes is negatively affected by the presence of both high and low glucose levels.

5. Multiple organ failure

Multiple organ dysfunction syndrome is defined as the presence of potentially reversible altered organ function involving two or more organ systems in acutely ill patients, such that homeostasis cannot be maintained without medical intervention.

Some of the early attempts to catgorise the severity of surgical infection and the risk of mortality were based on the observation that patients dying after surgical infection often went through a clinical course which is characterized by sequential organ failure. This has been called the "Multiple Organ Failure syndrome.[63]

The incidence of multiple organ failure syndrome has found to be 7 to 22% in cases of emergency operation and 31 to 55% in patients with intraabdominal abscess or bacteroides bacteremia.

A study of 176 patients with peritonitis was conducted by Bohnen and associates. The study revealed that patients with organ failure had a remarkably increased

mortality rate of 76% than those without. Delay in surgical intervention was associated with the development of multiple organ failure. Among those patients who presented with organ failure, those who did not undergo surgery within 24 hours of onset of peritonitis had an 88% mortality.[58]

Prognostic factors

Do we need scoring systems?

The complex nature of surgical infections, the multifaceted aspects of treatment, and the complexity of ICU support make evaluation of new diagnostic and therapeutic advances in this field very difficult. Scoring systems that provide objective descriptions of the patient's condition at specific points in the disease process aid our understanding of these problems

The success of TNM staging for Cancer, Glasgow coma scale for head injury and acute trauma score (ATS) for trauma has prompted researchers to look for scoring system in determining the outcome of disease with regard to peritonitis. The commonly tried scoring systems are:

1. Mannheim peritonitis index
2. Sepsis score of Elebute and Stoner
3. APACHE II score.

All the systems are mainly used to predict death in patients with surgical infections. Most of the scoring systems are inappropriate for use in therapeutic

decisions concerning individual patients.

In a country like India, where most of the critical care measures are unavailable and unaffordable by average citizens, it is vital that a scoring system should be evaluated which not only prognosticate accurately the outcome, but should also be simple and cost effective.

Mannheim Peritonitis Index (MPI)

MPI was originally devised from a study conducted in 1253 patients with peritonitis by Wacha et al[3]. The study was conducted between 1963 and 1979. A total of 20 factors which affect the prognosis of the patients were considered.

8 out of the 20 factors were found to be of significance in determining the prognosis of patients with peritonitis.

The information is collected at the time of admission and first laparotomy.

Each risk factor is assigned a score based on its influence in determining the outcome and a final score is arrived at. The maximum possible score by applying MPI index is 47. Those patients who had score more than 26 were deemed to be at high risk for mortality.

Risk Factors	Weighting if present
1. Age > 50 years	5
2. Female Sex	5
3. Organ Failure	7
4. Malignancy	4
5. Preoperative duration of peritonitis > 24 hr.	4
6. Origin of sepsis not colonic	6
7. Diffuse generalized peritonitis	6
8. Exudate Clear Cloudy, Purulent Faecal	 0 6 12

Definitions or organ failure

Kidney	Creatinine level ≥ 177 μ mol/l Urea level ≥ 167 m mol/l Oliguria < 20 ml / h.
Lung	PO_2 < 50 mm Hg PCO_2 > 50 mm Hg
Shock (definition according to Shoemaker)	Hypodynamic or Hyperdynamic
Intestinal obstruction (only if profound)(	Paralysis ≥ 24 h or complete mechanical ileus.

PO_2, Partial pressure of O_2, Pco_2, Partial pressure of CO_2

Detailed study of MPI was done by A. Billing [80] in 7 different centers and their data compared. They considered patients of perforated or postoperative peritonitis, peritonitis caused by pancreatitis, appendicitis and mesenteric ischemia for study. Fugger et al, divided patients into three groups based on their MPI score. Patients were classified as having scored less than 21, between 21 and 29 and those with score greater than 29. Those with score of less than 21 had the least risk for developing morbidity and mortality, whereas those with score greater than 29 had a

high mortality chance. Patients with score between 21 and 29 were designated as having intermediate risk.

Advantage of MPI

• It is easily applicable

• It allows for intra operative risk assessment

• Surgeon can know about the possible outcome and the appropriate management can be decided.

Patient with less score can be treated with minimal risks, while patient with high score may need aggressive approach with critical care monitoring. Concept of programmed relaparotomy, zip technique surgery may need to be considered in these cases. It is peritonitis specific index. Other scores like Apache-II score are not specific for peritonitis.

Disadvantages

- It is a one time score; hence post-operative complications may hamper the results.

- Peritonitis due to colonic perforation was deemed to be of low risk. Since most of the colonic perforations are usually secondary to malignancy, this may not be applicable uniformly.

MANAGEMENT

The most important aspect of treatment is that there should be no delay in the initiation of treatment as soon as a diagnosis of peritonitis is made. The treatment protocol can be classified as preoperative, operative.

Pre – Operative

Preoperative care consists of resuscitation, general support and antibiotic therapy. A study reviewed that it is preferable to delay surgery for a period of about two to three hours if the patient's general condition is poor and is haemodynamically unstable, during which time resuscitative measures may be carried out.

1. Analgesia.

Adequate analgesia is essential to make the patient comfortable. But it is advisable to delay the administration of analgesics until a diagnosis is made.

2. Haemodynamic monitoring.

There should be continuous monitoring of the vital signs. If necessary, arrangements for recording central venous pressure should be made.

3. Gastric Intubation.

A nasogastric tube of preferably large calibre should be used to evacuate the gastric contents and decompress the GI tract. Oral intake is not allowed.

4. Urinary catheterization

Bladder catherterisation serves to a keep a check on the urine output. Hourly urine output measurement can be used as an indirect indicator of the circulatory status.

5. Fluid resuscitation.

In cases of peritonitis there is massive fluid sequestration into the third space. Therefore it is of paramount importance to maintain adequate hydration of the patient.

6. Oxygen support

Patients with peritonitis may require ventilator support to combat hypoxia and acidosis.

7. Renal support

Patient should be hydrated adequately to prevent pre renal failure. Infusion of dopamine at renal doses may be necessary to increase perfusion to renal capillary bed.

8. Circulatory support

Inotropes such as dopamine and dobutamine may be necessary. But they must be administered only after adequate volume replacement is given and acidosis has been corrected.

9. Antipyretics

Anaesthetic complication increases in the presence pyrexia, especially when the core body temperaue is greater than 38.5°C. Effective antipyretic agents should be used to control the temperature.

Antibiotic Therapy

Though the primary measure for infection control is surgical "purge" of the peritoneal cavity, antibiotics have an important role in preventing late complications. It also essential for the controlling local spread of the intraperitoneal infection and bacteremia. [64]The antibiotic regimen that is chosen should cover gram positive, gram negative and anaerobic microorganisms.

Both single drug regimen with a broad spectrum antibiotic and multi drug regimen have tried in the management of peritonitis. No obvious difference have been found in the outcome of patients. [65]

Antibiotics used in single drug regimen are piperacillin/ tazobactum, ticarcillin/ clavulinic acid, imipenem/cilastin, meropenem, ertapenem, tigecycline.

For multidrug therapy, the following combinations are used : ciprofloxacin and metronidazole, aminoglycoside and clindamycin, third or fourth generation cephalosporin and metronidazole , aztreonam and metronidazole.

Effective antibiotic regimen has been found to improve the prognosis in cases of peritonitis.[66]In cases of patients who had been hospitalised for a long duration, pseudomonas coverage is adviced.

Immunosuppressed patients, patients who are on steroid therapy and those who have been receiving antibiotics for a long duration are at increased risk of candidal infection. Candidal infection was also commonly seen in peritoneal fluid cultures obtained from patients who are critical ill. Hence antifungal prophylaxis should be considered in these [67]

Studies have been conducted which have suggested that patients be changed over to oral antibiotics once they have been started on oral feeds. [68]

No standard protocol has been devised for the appropriate duration of treatment with antibiotics. The usual course of treatment is for around seven days. Depending upon the clinical scenario, patients may require prolonged therapy. Antibiotics are generally discontinued once there is no further clinical evidence of infection.

OPERATIVE MANAGEMENT

Source Control

Surgical management of peritonitis depends on the nature and location of the pathology. In cases of diffuse peritonitis, the dictum is to go for a midline incision, which makes it easier to identify the cause as well as to give proper lavage of the peritoneal cavity.

OPERATIVE PRINCIPLES:

1. Control of source of infection- Repair/Plug

2. Purge- Peritoneal lavage and toilet i.e. evacuate bacterial inoculums, pus and adjuvant.

3. Decompress- Treat or avoid intraabdominal compartmental syndrome.

4. Control- Prevent or treat persistent and recurrent infect ion [69]

PRINCIPLE – 1 REPAIR:

The surgical options available for source control, in frank gastrointestinal tract perforation are closure or exclusion or resection of the diseased viscous and the decision depend on the specific organ and pathology. Resection is the best option when feasible especially in inflammation without frank perforation in cases where disease progression is expected due to appendicitis, small bowel necrosis or

cholecystitis. In case of self-limiting inflammatory processes such as Crohns disease treatment is directed towards the underlying pathology and primary excision is not indicated.

Resection, though ideal, may not always be feasible. Certain technical factors may preclude resection, for example, in perforated extra peritoneal viscera such as duodenum and rectum due to the difficulty in mobilization. In such cases resection is usually the last option. Extremely intense inflammation may prevent safe excision in normally resectable organs example acute cholecystitis or acute perforated diverticulitis.

The management of left colonic perforation is controversial. Previously resection of the perforated segment, exteriorization of the proximal end as an end colostomy and distal mucous fistula or simple closure of the distal end (Hartmann's procedure) was done with restoration of intestinal continuity two to three months 1ater. The reason was, a high risk of anastomotic dehiscence was anticipated partly due to proximal faecal loading in case of an unprepared bowel. Several studies demonstrate safety of primary anastomosis when an intracolonic bypass device is used temporarily to rapidly carry colonic luminal contents past the anastomotic site with minimal contamination during the healing process

PRINCIPLE-2 PURGE:

A reduction in the bacterial count is achieved by aspirating all gross purulent exudates and by gently opening and debriding any loculations in the pelvis, paracolic gutters and subphrenic regions. Particulate debris should be removed with suction or moist swabs.

Intraoperative saline lavage augments mechanical debridement of particulate matter; the addition of antibiotics or antiseptic agents has no role. Saline acts as an adjuvant by impairing phagocytosis and leukocyte migration.

Price first advocated washing the contaminated peritoneal with large volumes of irrigant in 1905. In 1906, Torek reported that large volume irrigation reduced mortality in generalized peritonitis following appendicitis in 14%.

There are 3 basis principles of peritoneal lavage

1. To wash the digestive enzymes, that might have leaked into the peritoneal cavity.

2. To remove material like pus, blood and faeces that could harbor or nourish bacteria

The majority of surgeons lavage until the fluid is clear and use more than 1 l.

Continuous post operative peritoneal lavage has been recommended by some authors. At the conclusion of initial laparotomy multiple catheters or closed suction

drains are left in situ. Lavage is initiated in the immediate postoperative period using large exchange volumes (more than 2 liters) over three hour periods. Lavage is continued for 48-72 hrs or until the effluent is clears. Antibiotics and low dose heparin may be added to the lavage fluid. However there are no studies establishing the efficacy of this technique

PRINCIPLE-3 DECOMPRESSES:

During acute peritonitis more than 10 liters of inflammatory fluid may accumulate in the peritoneum and its sub-mesothelial loose connective tissue. The co-existent paralytic ileus, fluid accumulation in the peritoneal cavity, post resuscitation visceral and parietal edema increases the intraabdominal pressure producing a compartment syndrome. In this situation, if the abdomen is closed with tension, there will be impairment of cardiovascular, respiratory, renal and hepatic functions and also splanchnic blood flow and oxygenation. The answer to this problem lies in open abdomen or staged abdominal repair (STAR).

PRINCIPLE-4 CONTROL:

This principle aims at having control over the intra-abdominal processes like anastomotic healing, proper closure of perforation, and viability of bowel segments and formation of pus inside the abdomen. This aim is not achieved by the standard operation. This principle allows for frequent re-exploration and peritoneal toilet if required.

NEW OPERATIVE METHODS:

In 1993, the "International society of surgery" called several experts in this field to the "International surgical week" held at Hong Kong and decided on four basically different methods.[69]

OPEN ABDOMEN (LAPAROSTOMY):

This is defined as laparotomy without re-approximation and suture closure of abdominal fasciae and skin. Abdominal cavity is left open like an open wound and dressed and finally heals by granulation. This method takes care of principles-repair, purge and decompression. The disadvantages are, there is no control over intraabdominal process, exposed viscera may perforate and huge ventral hernia results since definitive closure is not possible. Hence it has lost its popularity.

COVERED LAPAROSTOMY (COLA):

This is defined as laparotomy without re-approximation and suture closure of abdominal fasciae and covering the facial gap with materials like merles or vicryl mesh. The viscera may also be covered with skin with relaxing incision.

PLANNED REPAPAROTOMY (PR):

In this approach abdomen is left open initially and re-explored at an interval of 12-24 hours for irrigation, debridement etc. Devices used to ease re-exploration include commercially available Zipper, Ethizip, Velcro, artificial burr, PTFE mesh

(Gortex) etc. this procedure allows for having control over intra-abdominal processes.

STAGED ABDOMINAL REPAIR (STAR):

This is a series of planned abdominal operations with staged re-approximation and final suture closure of the abdominal fasciae. It is planned either before or during the first operation called Index Star. The abdomen is closed temporarily with devices like Zip, Velcro etc. and controlled tension is exerted to the fascia. Re-laparotomies are performed at 24 hour intervals at operating room. Once problem is solved abdominal cavity is formally closed.

INDICATIONS FOR STAR: It is indicated in the following conditions:-

1. Diffuse peritonitis in critical patient condition.
2. Severe peritoneal edema.
3. Source of infection is not controlled.
4. Incomplete debridement of necrotic tissue.
5. When viability of bowel is uncertain, anastomosis/repair needs re-inspection
6. Uncontrolled bleeding with packing.
7. Infected pancreatic necrosis.
8. Massive abdominal wall loss.
9. Any intra-abdominal problem that is difficult or impossible to manage with a single operation.

ADVANTAGES OF STAR:

Staged abdominal repair technique allows for complete repair, debridement and purge. Anastomotic healing is monitored and any complications diagnosed early & corrected. Intra-abdominal compartment syndrome and its consequences are prevented. With the STAR technique colostomies and abdominal drains with their disadvantages are avoided. Finally this technique allows for suture closure of abdomen with sound healing.

Surgical Options for Common Causes of Peritonitis[36]

Perforated duodenal ulcer

The proper management is simple closure of the perforation using an omental patch (Graham patch). The addition of a definitive ulcer operation can be considered in patients who have had a perforation for less than 24 hours, are hemodynamically stable, with minimum peritoneal soiling and have no obvious comorbidities that will limit the safety of an extended operation. Definitive ulcer surgery is especially to be considered in those patients that have a history of chronic peptic ulcer disease.

Perforated gastric ulcer

All gastric ulcer should be biopsied. Ulcers on the greater curvature and high in the gastric fundus are commonly managed by wedge resection of the stomach in

order to simultaneously close the perforation as well as take biopsy of the lesion. If it is clearly representative of a benign perforation, a patch can be applied for closure especially if it is present in a peptic area such as the prepyloric region.

Small bowel infarction or perforation

Resection of the small bowel with primary anastomosis is the treatment of choice. Primary anastomosis may not be possible, if there is a high degree of peritoneal soiling, if the inflammatory response is severe, or the general condition of the patient is very poor. In such cases resection of the bowel with exteriorization of the ends is an option.

Appendicitis

Appendicectomy is the procedure of choice. In acute appendicitis laparoscopic appendicectomy has no significant complications. Conversion rate to open appendicectomy is in the range of 7-12.5% and frequently due to the presence of perforation of the appendix or an inflammatory mass which are commonly the cause of peritonitis. In case of appendicular abscess the abscess can be drained surgically by incising directly over the abscess on the abdominal wall. Transrectal drainage can be established if the mass is palpable on rectal examination. Alternatively ultrasound guided aspiration can be done.

Large bowel perforation

In perforation of diverticulitis resection of the perforated segment with end colostomy with a distal mucous fistula, followed by copious irrigation of the peritoneal cavity is the appropriate treatment. Restoration of continuity is delayed for 2-3 months.

In case of perforation of carcinoma the optional treatment is, resection, end colostomy with creation of a mucous fistula of the distal end or Hartmann's procedure (simple closure of the distal end). Resection with primary anastomosis as a single stage procedure may be attempted using a colonic bypass device or on table lavage of the proximal bowel.

Postoperative peritonitis

Re-anastomosis or closure of the anastomotic dehiscence is unlikely to be successful. In such cases options are either exteriorisation of the anastomotic ends where possible, as in the colon, small bowel, or stomach, or if exteriorisation is not possible, as in the case of retroperitoneally fixed duodenum deliberate control iatrogenic fistula formation or in the case of distal retroperitoneally fixed bowel defunctioning of that part of the bowel with drainage should be done.

Cholecystitis with perforation or pericholecystic abscess

Cholecystectomy is the procedure of choice and laparoscopic cholecystectomy

is the gold standard for treatment of cholecystitits. Tube cholecystostomy is used only when the patient is critically ill or is an unsuitable candidate for general anaesthesia.

Intra-abdominal abscess

Traditionally surgical drainage has been done, but presently optimal patient outcome is reported with ultrasound or computed tomograpy guided percutaneous drainage. Subsequently in cases such as appendicitis or cholecystitis definitive surgery may taken up. Percutaneous drainage helps to avoid surgery in case of perianastomotic abscess or it may help to simplify the surgical approach as in peridiverticular abscess where delayed resection with primary anastomosis can be done.[49]

Laparoscopy represents a major recent advance in the diagnosis and management of acute abdomen. Several studies demonstrate the superiority of laparoscopic cholcystectomy in the setting of acute cholecystitis with one series, reporting a conversion rate of twenty seven percent in the emergency setting as against five percent in the elective setting. Laparoscopic appendicectomy does not have any significant complications and conversion rate are 7% -12.5% and are due to appendicular perforation or mass formation. Laparoscopic management of perforated gastric and duodenal ulcers is a viable option.[70]

COMPLICATIONS OF PERITONITIS

It can be due to peritonitis itself and post-operative:

1. Septicaemia

2. Bacterial Toxaemia

3. Electrolyte imbalance

4. Acute intestinal obstruction due to peritoneal adhesions.

5. Residual abscess

i. Pelvic abscess.

ii. Subphrenic abscess

6. Paralytic ileus

7. Renal failure

8. Cardiac failure

9. Pulmonary complications.

a. Bronchitis.

b. Atelectasis.

c. Pneumonia

d. Pulmonary embolism

e. Bronchopneumonia.

10. Deep vein thrombosis.

11. Burst abdomen.

MATERIALS AND METHODS

MATERIALS AND METHODS

The study is done in 50 patients with peritonitis due to hollow viscous perforation who presented to **PSG Hospitals, Coimbatore,**

The study is a clinical, prospective, observational and open study.

METHOD OF COLLECTION OF DATA

The study is done after obtaining a detailed history, complete general physical examination and systemic examination.

The patients are subjected to relevant investigations like x-ray erect abdomen, CXR, USG and routine investigations like Hb, TC, urea, creatinine, serum electrolytes.

All investigations and surgical procedures were carried out with proper informed written consent .

The data regarding patient particulars, diagnosis, investigations, and surgical procedures is collected in a specially designed case recording form and transferred to a master chart. The data is subjected to statistical methods like mean, proportion, percentage calculation and wherever necessary chi square test for proportion are used.

INCLUSION CRITERIA

- Age > 15 years
- Diagnosed to have peritonitis and on whom surgical intervention is planned

EXCLUSION CRITERIA

- Conservatively managed patients – pancreatitis , spontaneous bacterial peritonitis, patients on peritoneal dialysis
- Abdominal injuries with associated solid organ or vascular injuries.
- Polytrauma patients
- Peritonitis secondary to anastomotic leak

MODE OF STUDY

The detailed history and proper clinical findings were entered in a case recording form. Patients were subjected to methodical physical examination to assess their general condition. Local examination of abdomen was done and relevant findings were recorded. Rectal examination was done in all cases,

The required and routine investigations were done to establish the diagnosis.

Preoperatively all patients received supportive treatment for correction of hypotension and electrolyte abnormalities. During laparotomy, intra-abdominal examination of all organs was made in addition to the specific pathology.

MPI scoring was done in all patients and patients were classified as those with score less than 21, between 21 to 29, and more than 29.

The nature of surgical procedure was planned preoperatively based on the suspected pathology and the general condition of the patient. But the final choice of the procedure was decided upon the merit of each case and the intra operative finding. The issue of placing a drain in the peritoneal cavity was left to the discretion of the operating surgeon. Post operative period was monitored; intake output charts and vital charts were maintained.

Patients were followed up for a period of one month post surgery to assess for development of complications

OBSERVATION AND RESULTS

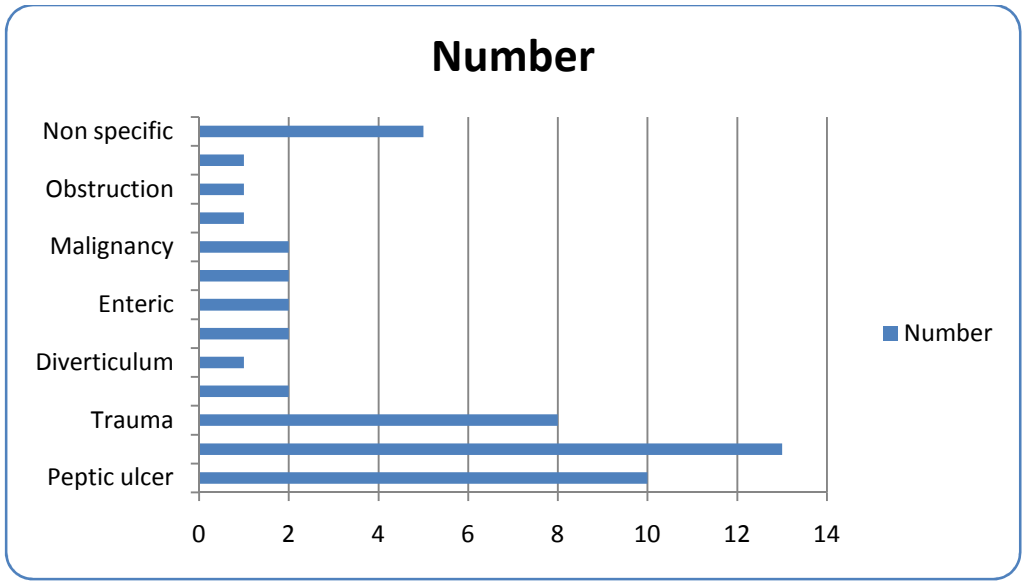
OBSERVATION AND RESULTS

The study was conducted in a population of 50 patients who had been diagnosed as having peritonitis secondary to hollow viscus perforation.

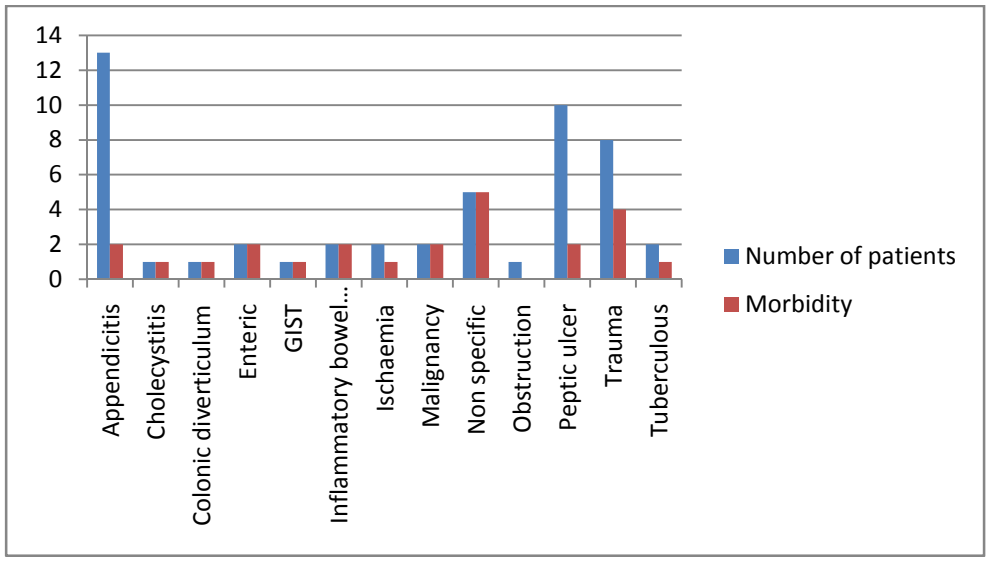
ETIOLOGICAL FACTORS

Etiology	Number of patients	Percentage	Morbidity	Avg mpi score
Appendicitis	13	26	2	13.0769
Cholecystitis	1	2	1	20.0000
Colonic diverticulum	1	2	1	21.0000
Enteric	2	4	2	20.5000
GIST	1	2	1	18.0000
Inflammatory bowel disease	2	4	2	21.0000
Ischaemia	2	4	1	24.5000
Malignancy	2	4	2	22.5000
Non specific	5	10	5	25.2000
Obstruction	1	2	0	31.0000
Peptic ulcer	10	20	2	17.9000
Trauma	8	16	4	18.2500
Tuberculous	2	4	1	22.5000
Total	50	100	24	18.6600

It was observed that perforated appendix was most common cause of peritonitis in our study accounting for 26 percent of the cases. This followed by perforation of peptic ulcer which was 20 % of the cases. Trauma was found to be a significant cause of gastrointestinal perforation accounting for 16 % of the cases in our study. Enteric illness, inflammatory bowel disease, ischaemia, malignancy and tuberculosis were each found to constitute 4 per cent of the cases. Cholecystitis, colonic diverticulum, GISTs, perforation following bowel obstruction, each formed 1 percent of the cases. No identifiable cause of perforation could be found in 10 percent of the cases.



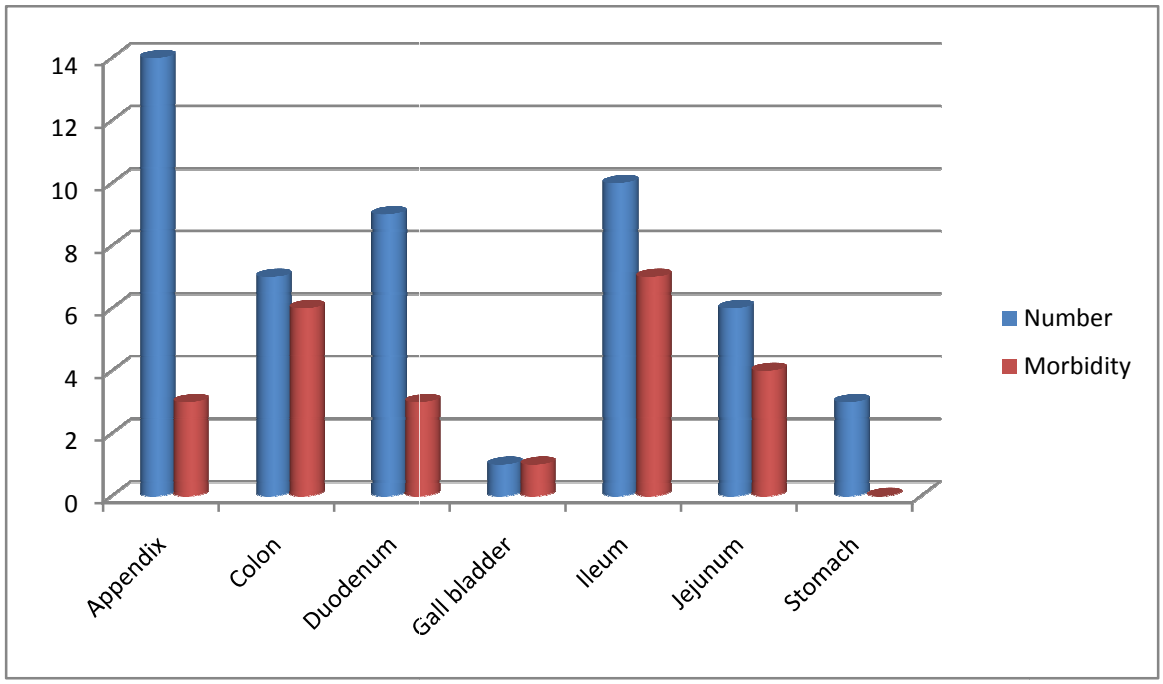
It was found that though perforated appendix and peptic ulcer were the most common etiologies, they were both associated with low morbidity rates. Perforated appendix had a morbidity rate of 15 per cent and perforated peptic ulcer a morbidity rate of 20 %. Likewise they were also associated with a lower average MPI score. The average MPI score in cases of perforate appendix was 13.07 and the average in cases of perforated peptic ulcer was 17.9.



SITE OF PERFORATION

Site of perforation	Number	Percentage	Morbidity	Avg MPI score
Appendix	14	28	3	13.5000
Colon	7	14	6	23.8571
Duodenum	9	18	3	17.7778
Gall bladder	1	2	1	20.0000
Ileum	10	20	7	22.5000
Jejunum	6	12	4	20.8333
Stomach	3	6	0	15.6667
Total	50	100	24	18.6600

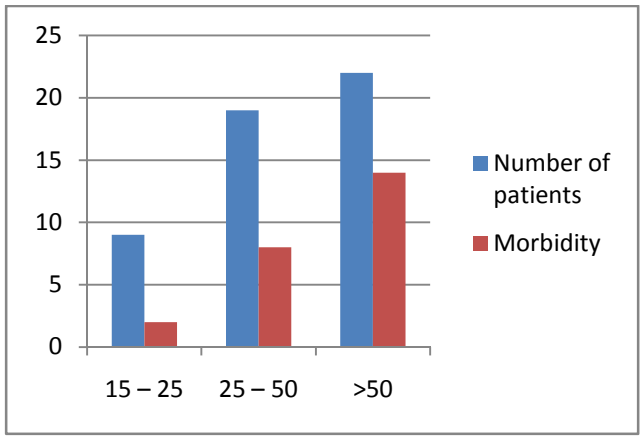
The most common site of perforation was the appendix. 28 percent of the patients in the study had come with appendicular perforation. 21.42 percent of appendicular perforations had morbidity. Perforation involving the ileum was found to be the next most common, accounting for 20 percent of the cases. The average MPI score for the ileal perforations was 22.5 and the morbidity rate was 77.77 percent. Duodenal perforations were the third most common at 18 percent. Duodenal perforations were to have a lower morbidity rate of 33.33 percent and had an average MPI score of 17.77.



AGE DISTRIBUTION

Age group	Number of patients	percentage	Morbidity	Avg MPI score
15 – 25	9	18	2	14.6667
25 – 50	19	38	8	17.2632
>50	22	44	14	21.5000
Total	50	100	24	18.6600

The study was conducted in patients over 15 years of age. It has been found that perforation peritonitis is more common among the elderly population. 44 percent of cases occurred in patients who were aged 50 and above. 38 per cent of cases were seen in the middle aged (25-50 years of age). Only 18 percent of cases were seen to occur in the age group of 15-25 years.



It is seen that with increasing age, there is an increase in the morbidity rate. The morbidity rate is 22.22 % in the age group of 15 to 25 years, 42.1 per cent in the age group of 25 to 50 years, and 70 percent in patients over 50 years of age. Both the mortalities that occurred in the study was in persons over 50 years of age. The p value is 0.05.

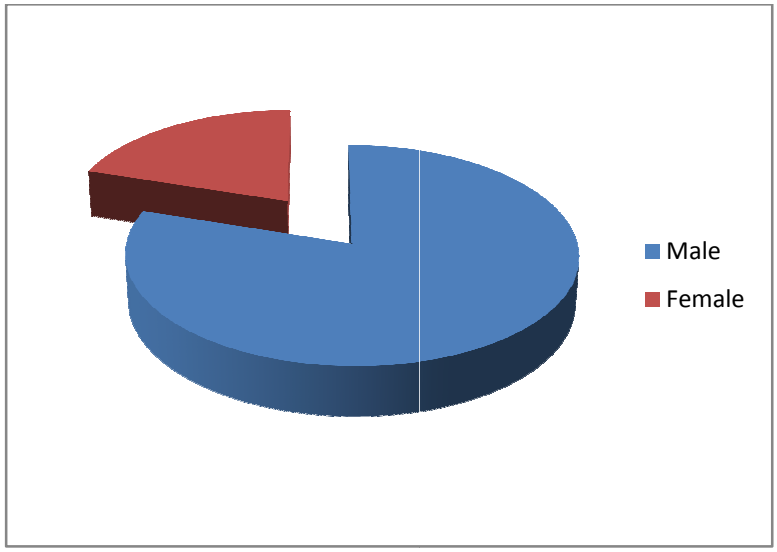
The average MPI score also shows an increase with increasing age. The average MPI scores for the age groups 15-25 years, 25 -50 years and >50 years were found to be 14.66, 17.26 and 21.50 respectively.

SEX DISTRIBUTION

Sex	Number	Percentage	Morbidity	Avg MPI score
Male	40	80	21	18.4500
Female	10	20	3	19.5000
Total	50	100	24	18.6600

80 percent of patients in the study were found to be males. Females accounted for 20 percent of the cases.

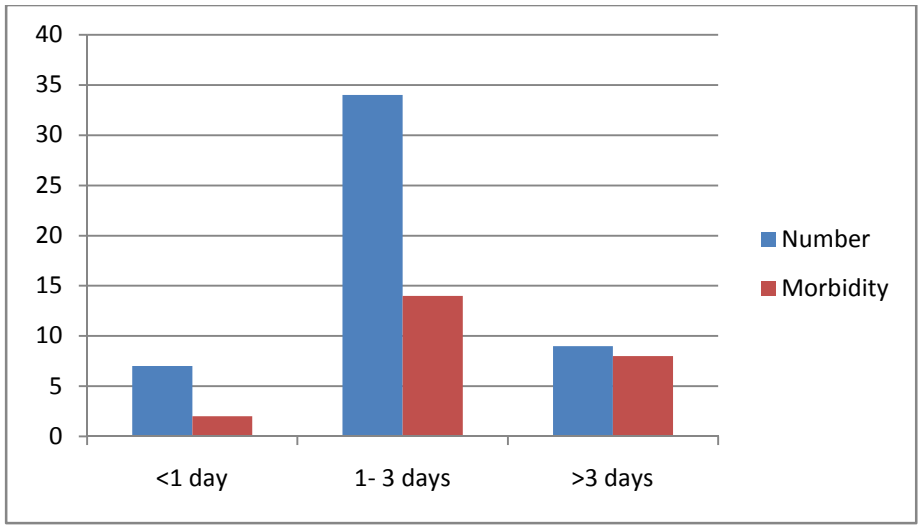
The morbidity rate in men was found to be 53.84 percent where as in women it was 30/33.33 percent. But the average MPI score was found to be greater in women (19.5) as compared to men (18.45). However this was found not to be significant. The p value is .310. One of the patients who had died during the study was male and the other female.



DURATION OF SYMPTOMS

Duration of peritonitis	Number	Percentage	Morbidity	Avg MPI score
<1 day	7	14	2	13.2857
1- 3 days	34	68	14	18.5882
>3 days	9	18	8	23.1111
Total	50	100	24	18.6600

Fourteen percent of patients presented within a day of onset of symptoms. These patients had a morbidity rate of 28.5 percent and the average MPI score for these group of patients was 13.2. 68 percent of patients presented within 24 to 72 hours after onset of symptoms. The morbidity rate in these patients was 43.75 percent and the average MPI score 18.58. The percentage of patients who presented after 72 hours was 18. These patients had a morbidity rate of 88.88 percent and the average MPI score was higher than the other two groups at 23.11. Both patients who had died during the study presented 3 days after onset of symptoms.

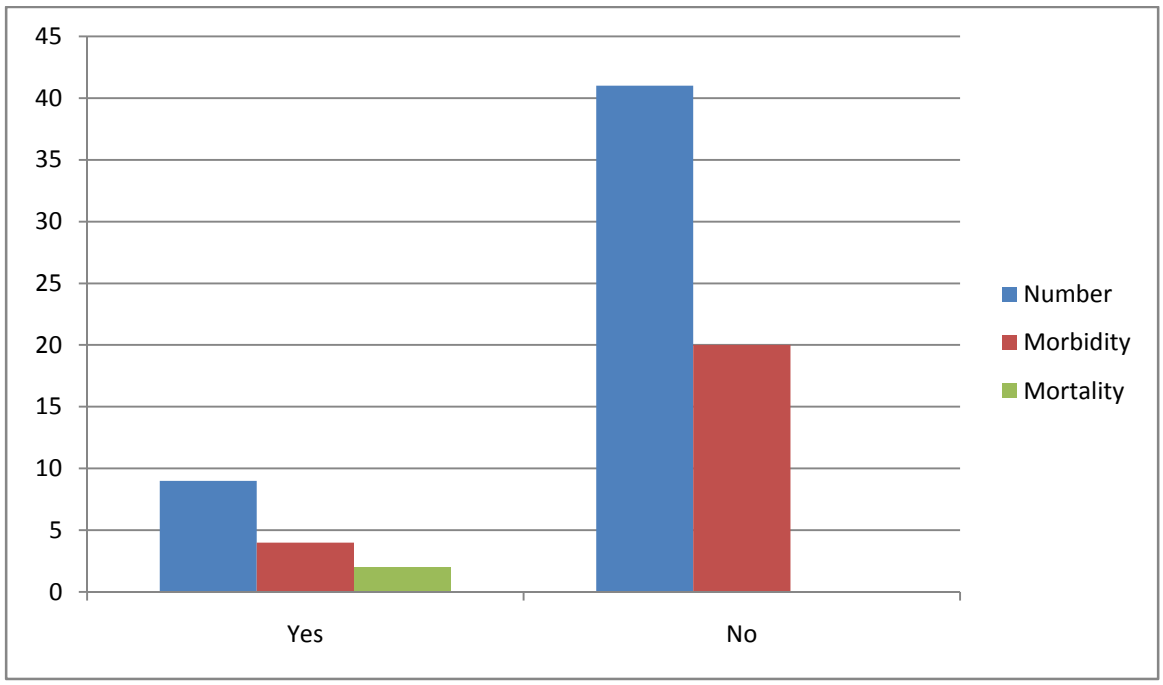


ORGAN FAILURE AT PRESENTATION

Organ failure at presentation	Number	percentage	Morbidity	Mortality	Avg MPI score
Yes	9	18	4	2	25.0000
No	41	82	20	0	17.2683
Total	50	100	24	2	18.6600

In the study population 18 percent presented with organ failure at admission. These patients had a morbidity rate of 57.14 % and average MPI score of 25. Both the patients who had mortality in the study presented with organ failure on admission.

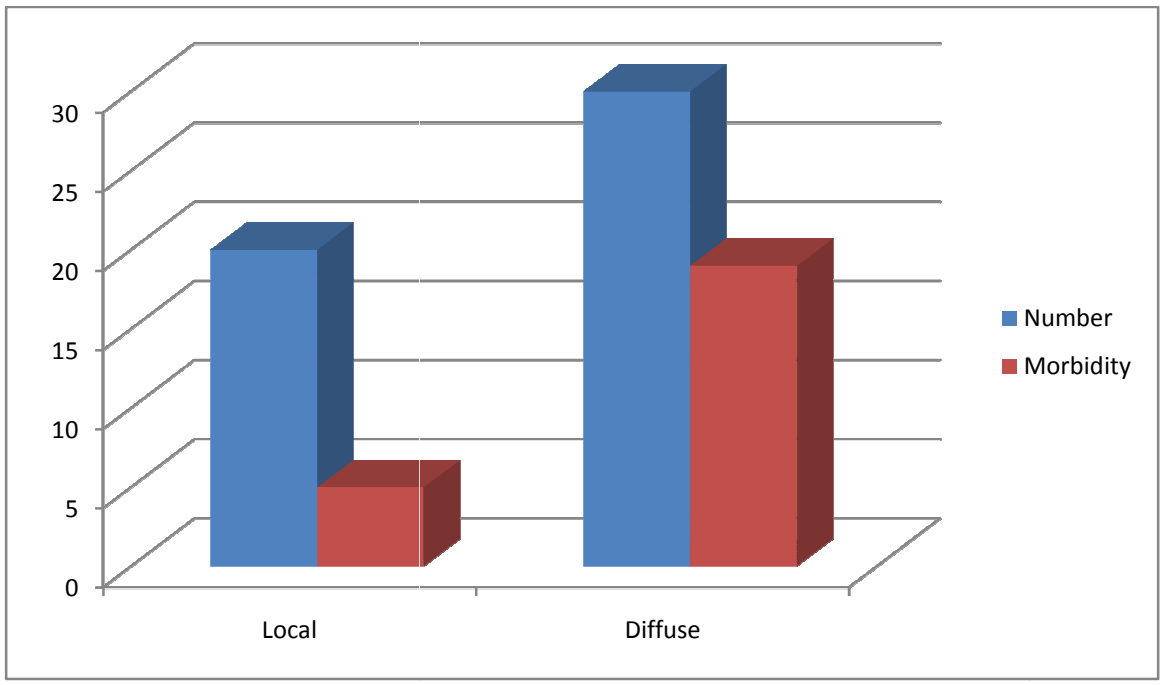
82 percent of patients did not have any organ failure at the time of presentation. The patients had a morbidity rate of 48.78 and the average MPI score was 17.26. The p value is 0.008.



EXTENT OF PERITONITIS

Extent of peritonitis	Number	Percentage	Morbidity	Avg MPI score
Local	20	40	5	14.6500
Diffuse	30	60	19	21.3333
Total	50	100	24	18.6600

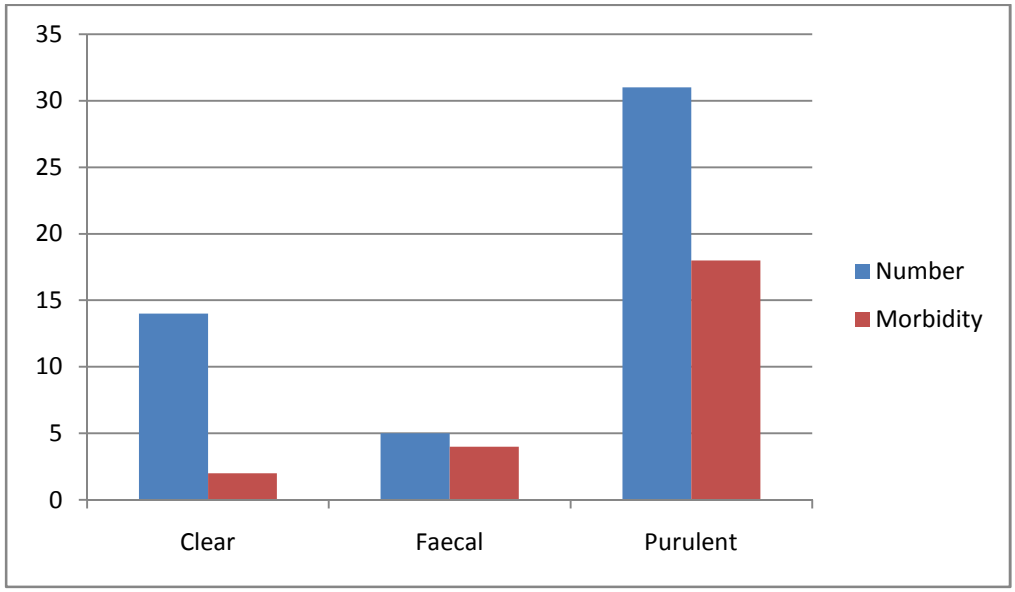
In our study, it was found that 40 percent of the patients presented with localised peritonitis. The patients had a morbidity rate of 25 percent and a low average MPI score of 14.65. 60 percent of patients came with generalized peritonitis. These patients had a higher average MPI score of 21.33 and the morbidity rate was 67.85 percent. The p value is 0.007



TYPE OF PERITONEAL EXUDATE

Type of exudates	Number	Percentage	Morbidity	Average MPI score
Clear	14	28	2	16.0714
Faecal	5	10	4	27.800
Purulent	31	62	18	18.3548
Total	50	100	24	

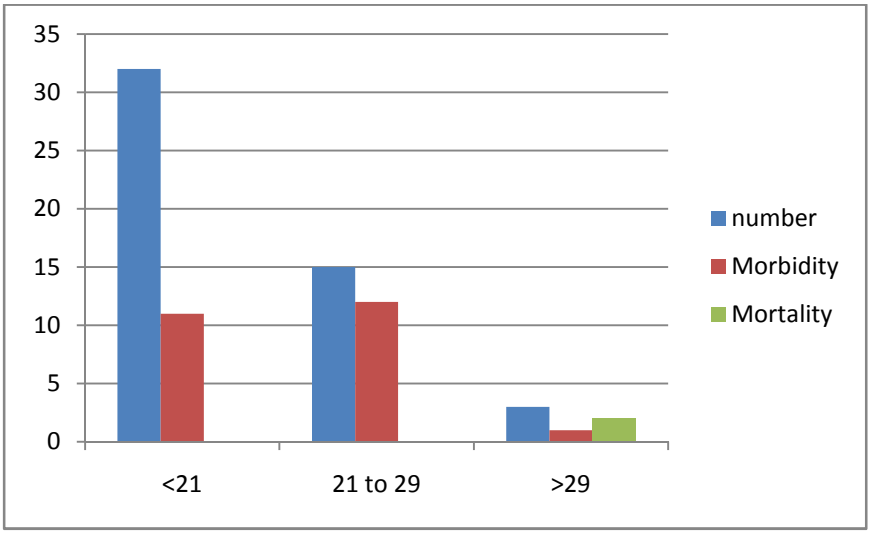
62 percent of patients in our study had purulent peritoneal fluid. These patients had a morbidity rate of 58.06 percent and an average MPI score of 18.70. 10 percent of patients had faecal peritonitis. These patients had a high average MPI of 27.80 and the highest morbidity rate. Twenty eight percent of patient had no pus or faecal contamination of the peritoneal fluid. These patients had a morbidity rate of 15.38 percent. The p value is 0.004.



MPI SCORING

MPI score	number	Percentage	Morbidity	Mortality
21 and less	32	64	11	0
21 to 29	15	30	12	0
29 and more	3	6	1	2
Total	50	100	24	2

In finality, it was found that 64 percent of patients had MPI score of less than 21. These patients had a morbidity rate of 34.37. 30 percent had MPI score within 21 to 29. These patients had a morbidity rate of eighty percent. There were three patients who had MPI score of above 29. Two of these patients died and the remaining one had post op morbidity. The mortality rate was 66.66 percent in this group. There was no mortality in the other two groups. The association of increasing MPI score with mortality and morbidity is found to be significant.The p value is <0.001.



DISCUSSION

DISCUSSION

Peritonitis resulting from perforation of the gastrointestinal tract is a common surgical emergency in India.

Etiological factor:

In our study conducted at PSG hospitals in a group of 50 patients who have been diagnosed to have perforation peritonitis, it was found that appendicular perforation was the most common cause of perforation peritonitis. 13 patients out of the study population of 50 patients had appendiular perforation. The next most common was perforation due to peptic ulcer disease. Trauma was the third most common cause of perforation peritonitis.

In a study conducted by Jhoba et al, at Government Medical College and Hospital (GMCH), Chandigarh, it was found that the most common causes of perforation peritonitis were perforated duodenal ulcer, appendicitis and trauma in that order. In our study too, the top three common causes were found to be the same. [71]

Site of perforation

In our study it was found that appendicular perforation was the most common. This was followed by gastro duodenal perforation. Ileum was the third most common site of perforation. The causes for ileal perforations are varied. Enteric fever,

trauma, ischaemia, obstruction and tuberculosis were among the pathologies associated with ileal perforation. Also cases of non specific ileal perforation were also noted. It was found that appendicular and duodenal perforations had low morbidity rates. There were three cases of gastric perforation and none had any morbidity. On other hand ileal and colonic perforations carried high morbidity rates.

In a study conducted by Batra and others in central rural India, it was found that the most common site of perforation was gastro duodenal, followed by small bowel and appendicular.[72] Similar results were seen in study conducted by Ramachandran and others in Mysore.

In our study we found the top three sites of perforation to be similar. But appendicular perforations were found to be more common than gastro duodenal and small bowel perforation in our study.

Age distribution :

In our study it was observed that cases of perforation peritonitis were more in common in the elderly age group. Also it was noted that these patients had a higher mortality rate compared to patients of younger age. The average MPI score for patients over 50 years of age was also found to be higher than those patients less

than 50 years. The lowest morbidity rate was seen in patients between 15 to 25 years of age. The mortality rate in our study is to increase with increase in age. The average MPI score has a linear relationship with increasing age.

That increasing age is a poor prognostic factor in patient presenting with peritonitis is documented in numerous studies.

In a study conducted by Pisanu A and others, it was found that age is an independent risk factor in predicting the prognosis in patients with perforated colonic diverticulitis.[73]

In a study conducted by Chandrasekhar and others, it was found that age above 50 years is a poor prognostic factor in patients with perforative peritonitis[74]

In a study conducted by Mehmet and others, it was found elderly patients with peritonitis were at a higher risk for mortality.[75]

Sex distribution :

The MPI scoring system attributes a higher risk for the female sex. In our study, it was found that 80 percent of the patients were males and only 20 percent were females. The morbidity rate among male patients was found to be higher than in female patients. However this was found to be not significant statistically. The

digression could be from the fact that there were lesser number of female patients in the study.

Duration of symptoms :

In our study, it was found that those patients who presented themselves within 24 hours of onset of symptoms had the lowest morbidity rate. A majority of the patients in the study presented between 1 to 3 days after onset of the symptoms. It was found that the morbidity rate was higher with more delay in presentation. The morbidity rate is only 28.5 percent in patients presenting within 24 hours and increases to 88.88 percent in those patients who presented after 3 days. The rise in morbidity correlates with higher MPI score in those who have delayed presentation.

In a study conducted by Pisanu A and others, it was found that duration of symptoms is an independent risk factor in predicting the prognosis in patients with perforated colonic diverticulitis.[73]

In a study conducted by Chandrasekhar and others, it was found that duration of perforation is a poor prognostic factor in patients with perforative peritonitis.[74]

In a study conducted by *Ntirenganya and others, it was found that duration of symptoms more than 24 hours was adversely associated with the outcome in cases of peritonitis.*[76]

In a study conducted by Mehmet and others, it was found that symptom duration was an important factor in deciding the outcome of patients with peritonitis.[75]

In a study conducted by Ranju singh and others in New Delhi, it was inferred that delay in presentation led to poorer outcome in patients with peritonitis.[77]

Organ failure :

In our study, it was found that those patients who had organ failure at the time of presentation had a higher morbidity rate. There were two mortalities noted in the study. Both the patients had organ failure at the time of presentation. That the MPI scoring system accords a higher risk to those with organ failure seems justified.

In a study conducted by Basnet and others, done to assess the predictive power of Mannheim peritonitis index, it was found that the presence of organ failure correlated with a poorer outcome.[78]

In a study conducted by Chandrasekhar and others, the presence of shock at the day of presentation is associated with poorer prognosis.[74]

In a study conducted by *Ntirenganya and others, it was found that the presence of organ failure was adversely associated with the outcome in cases of peritonitis.*[76]

Extent of peritonitis :

Even though appendicular perforation was found to be the most common cause of perforation peritonitis in our study, it was found that the number of patients with diffuse peritonitis numbered more than those with localised peritonitis. The MPI scoring system attributes a higher risk to those with generalized peritonitis and likewise these patients were found to have a higher morbidity rate.

In a study conducted by Correia and others in Mexico to evaluate the efficacy of Mannheim Peritonitis Index in predicting death in oncologic patients, it was found that the presence of diffuse peritonitis resulted in bad prognosis. [4]

In a study conducted by Basnet and others, it was found that the presence of diffuse peritonitis correlated with a poorer outcome. [78]

Character of peritoneal exudate:

According to the MPI scoring system patients with faecal contamination had a poorer prognosis. In our study, we found this to be justified since patients with faecal peritonitis had a hundred percent morbidity rate. One of the mortalities in the study also had faecal contamination of the peritoneal cavity. The correlates with the fact that colonic perforations carried a high morbidity rate as compared to gastro duodenal perforations.

Patients with purulent peritoneal exudate had a higher morbidity rate than those with clear peritoneal exudate, justifying the scores accorded to peritoneal fluid exudates as per the MPI system.

In a study conducted by *Ntirenganya and others, it was found that the presence of faecal contamination was adversely associated with the outcome in cases of peritonitis.*[76]

MPI SCORE :

The Mannheim peritonitis index is a peritonitis specific index which is easily applicable. It is based on clinical parameters that are routinely assessed. It also

allows for intra operative evaluation of the patient to provide a better assessment of the final prognosis. Numerous studies have been done which have validated its accuracy and applicability in predicting the prognosis in patients with peritonitis. Higher Mannheim index score has a strong association with increased mortality.

Over the years, there has been a fall in the mortality rates in cases of peritonitis. This has been attributed to better intensive care support, a better understanding of the path physiology of the peritonitis. More appropriated surgical techniques have been devised in the management of peritonitis. In high risk cases, definitive procedures are deferred and the focus is on clearance of source of infection. The concept of staged laparotomy has gained popularity in recent times in the management of severely ill patients in whom reexploration is expected.

In our study a total of 50 patients with perforation peritonitis were followed. Only two mortalities were noted in the study. It has been found that the Mannheim peritonitis index has been a good predictor of mortality as well morbidity in patients with peritonitis. The patients were grouped as those having score less than 21, score between 21 and 29 and those with score greater than 29. It was found that morbidity rate was the least in those with scores less than 21. Patients whose score was between 21 and 29 had a higher morbidity rate, but no mortalities were noted

in this group. Those patients whose MPI score was more than 29 had the highest morbidity rate. Both the mortalities that occurred during the study had scored more than 29.

It was also found in our study that with the exception of sex based risk assessment, all other parameters of the Mannheim peritonitis index were closely associated with the prognosis of the patients.

Fugger and others conducted a retrospective study in 113 patients to test the prognostic value of Mannheim peritonitis index. They found the mortality was nil for patients whose score is less than 21 and 100 percent for those with score greater than 29. They concluded that the MPI score was highly accurate in the predicting the prognosis.[79]

Billing and others conducted a study in 2003 patients, in which the predictive of Mannheim peritonitis index was assessed. They found that the threshold index score of 26 was specific and sensitive in predicting the mortality. They also divided patients into three groups, those with score <21, 21-29 and > 29. They found the mortality rate to be lowest in the first group and highest in the last group with scores greater than 29. They concluded that the MPI was easy to apply

scoring system that was reliable and accurate in predicting the prognosis in patients with peritonitis[80]

Ermolov and others made a retrospective analysis in 100 patients with diffuse peritonitis. They found that the mortality was hundred percent in patients whose score was more than 29. No deaths were noted in those patients whose score was between 12 and 20.[81]

Kusumoto yoshiko and others conducted a study on 108 patients with peritonitis and tested the ability of Mannheim Peritonitis Index (MPI) in predicting the prognosis. They found that those patients with score less than 26 had a mortality of just 3.8% as compared to 41 per cent in patients whose score was more than 26. A comparison of MPI and mortality showed patients with a MPI score of 26 or less to have mortality of 3.8%, where as those with a score exceeding 26 had mortality of 41.0%.[82]

Gedik and others conducted a study in 96 patients with enteric perforation. They found that the Mannheim peritonitis index and the time interval till surgical intervention is started were risk factors which independently affected the morbidity in these patients.[83]

Qureshi AM and others analysed the efficacy of Mannheim peritonitis index in the 126 patients with secondary peritonitis. They found a significant association between increasing MPI score and mortality.[84]

In a study conducted by Pisanu and others to evaluate the factors that play a role in predicting mortality rates in patients with perforated colonic diverticulitis, it was found that the MPI was highly specific and sensitive in predicting the mortality with a score of greater than 27.[73]

In a study conducted by Chandrasekhar and others in 50 patients with perforative peritonitis in Karnataka, it was found that the MPI score accurately predicting morbidity and mortality rates in these patients.[74]

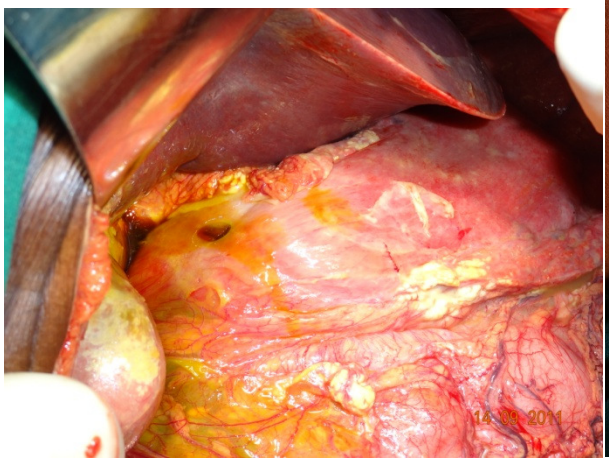
Notash and others conducted a study in eighty consective patients of secondary peritonitis who underwent similar surgical management to assess the efficacy of MPI score and multiple organ failure score. They concluded that both scores were equally effective in predicting the outcome.[85]

CONCLUSION

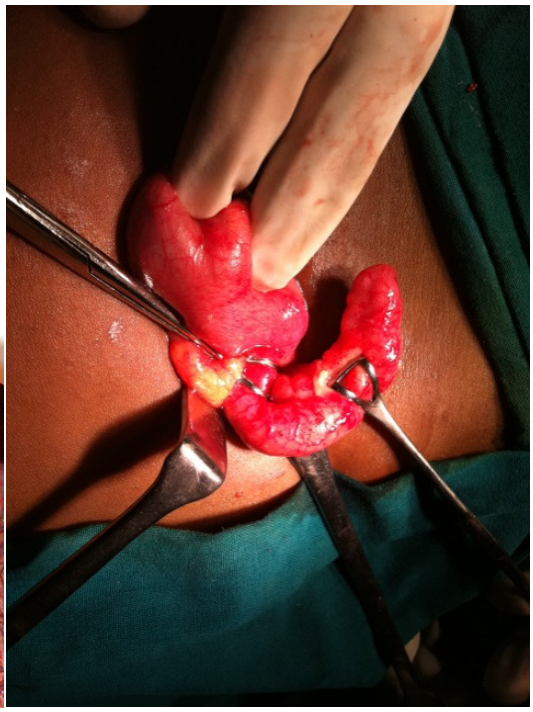
CONCLUSION

Despite advancements in the realm of medical science, the management of patients with peritonitis continues to be demanding. In our study it was found that appendicular perforation was the most common cause followed by gastro duodenal perforation. Trauma was found to be a significant cause of perforation peritonitis. It was found that ileal perforations constitute a major proportion of cases of secondary peritonitis and the causes of ileal perforations to be varied. More males were found to present with perforation peritonitis than women. It was found that perforation of the gastrointestinal tract is more common among the elderly and that these patient also have a poorer prognosis.

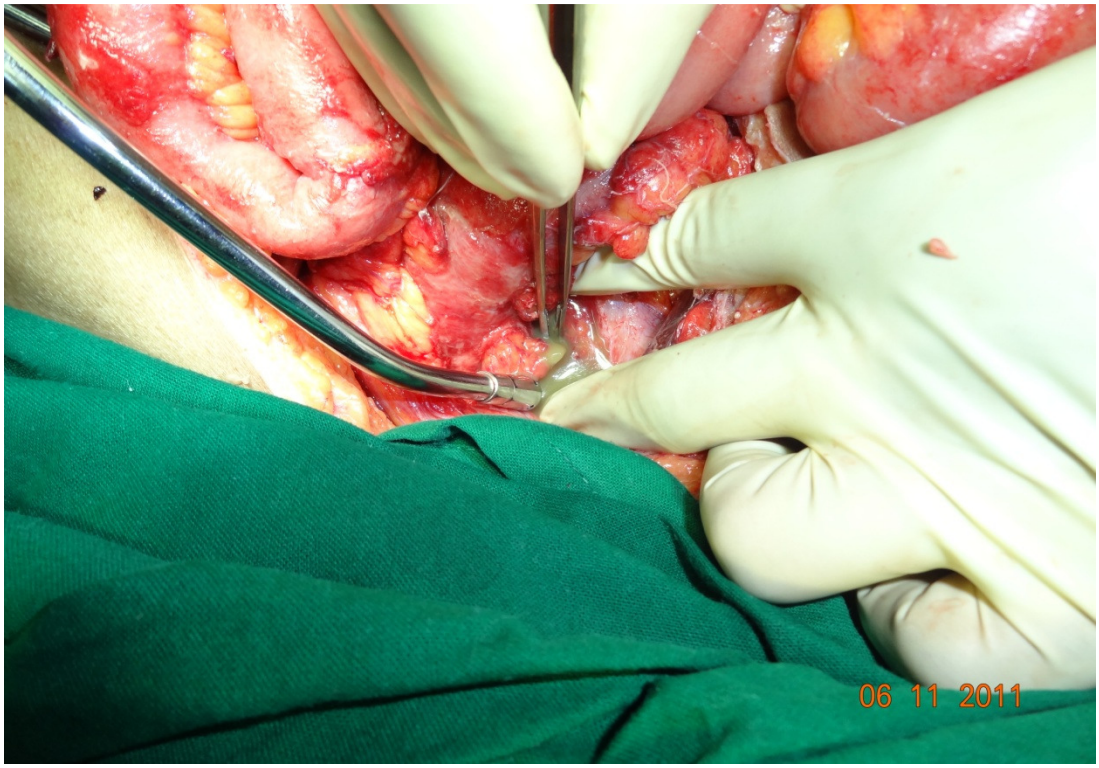
The mortality rate over the years have come down due to better supportive care and by implementing appropriate operating protocols in these patients. Nevertheless the challenges presented remain remarkable. A specific scoring system which is easy to apply, simple to calculate and accurate in prediction will be of great use in the management of patients with peritonitis. It has been found that the Mannheim peritonitis index duly fulfils these criteria. The individual parameters of the index with the exception of sex based risk assessment were found to positively correlate with the prognosis in our study.



4 DUODENAL PERFORATION



5 APPENDICULAR PERFORATION



6 RECTAL PERFORATION

7 SMALL BOWEL PERFORATION

8 WOUND DEHISCENCE

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APPENDIX

Name :
Age :
Sex :
Marital status :
Occupation :
Address :

I.P.No :
DOA :
DOD :

CHIEF COMPLAINTS
Pain abdomen :
Fever :
Vomiting :
Abdominal distension :
Bowel Disturbances :
Urinary Disturbances :
Loss of appetite
Loss of weight :
Any other :

PAST HISTORY
Similar illness :
Any history of surgeries :
Co Morbid illness:

D. FAMILY HISTORY
Malignancies :
Similar illness :

E.PERSONAL HISTORY
Smoking :
Alcohol :
Type of diet :
Any other habits :
Bowel habits :
Bladder habits :

F.DRUG HISTORY

G. MENSTRUAL HISTORY
Menarche :
Menstrual cycles :
Menopause :
Any other disturbances :

H. OBSTETRIC HISTORY

GENERAL PHYSICAL EXAMINATION
Built : Well / Moderate / Poor
Nourishment : Well / Moderate / Poor
Pallor : Mild / Moderate / Severe
Icterus : Mild / Deep
Pedal edema : Pitting / Non Pitting
Febrile : Yes / No
Dehydration : Yes / No
Gen. Lymphadenopathy : Yes / No

Pulse :
Rate
Rhythm
Volume

Blood Pressure :
Respiratory Rate:
SpO2:
GCS :

EXAMINATION OF ABDOMEN

1. INSPECTION
a) Shape : Flat / Scaphoid / distended
b) Any mass / fullness :
c) Umbilicus:
d) Visible veins:
e) Visible peristalsis :
f) Flanks :
g) Hernial orifices :
h) All quadrants moving equally with respiration
i) Scars : No / site / nature of healing
j) Sinuses : No / site / surrounding skin / nature of discharge
k) Fistula
l) Any others

2. PALPATION
Soft:
Guarding :
Rigidity : Localized / generalized
Tenderness : Present / Absent Rebound tenderness:
Palpable mass:
Organomegaly:

3. PERCUSSION
Free fluid :
Bladder : Yes / No
Renal angle : Normal / dull
Obliteration of liver dullness :

4. AUSCULTATION
Bowel sounds : Yes / No
Frequency
Character

P/R :
Wall
Lumen
Nature of finger stain

P/V :

RS :

CVS :

INVESTIGATIONS

a) Blood :

Hb% TC DC ESR

Blood group

FBS

Blood urea

Serum creatinine

ABG

Serum electrolytes

b) Urine : Sugar Albumin

Microscopy

c) Chest X-ray :

d) Plain X-ray abdomen:

e) Ultrasound :

f) CT abdomen (if done)

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CLINICAL DIAGNOSIS

OPERATIVE DATA:

FINDINGS:

Character of peritoneal fluid : clear / cloudy or purulent / faeculent

Peritonitis : generalised / localised

Site of perforation :

Etiology of perforation :

Type of procedure done :

Drain:

MPI SCORE -

- Age >50 years - 5
- Female sex - 5
- Organ failure - 7
- Preop duration of peritonitis >24 Hrs - 4
- Malignancy - 4
- Origin of sepsis : Non-colonic - 4
- Diffuse generalised peritonitis - 6
- Character of peritoneal fluid

Fecal - 12

Purulent - 6

Clear - 0

POST-OP-PERIOD

Complications:

Duration of hospital stay:

Status at the time of discharge

MORTALITY

Cause:

FOLLOW UP

MASTER CHART

s.no	IP NO:	Age	Sex	Duration of peritonitis	Organ failure at admission	Etiology of perforation	Site of perforation	Diffuse peritonitis	Type of exudate	MPI score	Morbidity	mortality
1	I12029569	81	M	2 days	Yes	Peptic ulcer	Stomach	no	Clear	20	Nil	No
2	I12027649	59	M	3 days	No	Peptic ulcer	Duodenum	Yes	Purulent	25	Wound infection	no
3	I12033616	27	F	3 days	No	Tuberculous ulcer	Ileum	Yes	Purulent	25	Nil	no
4	I12032513	60	M	4 days	No	Colonic diverticulum	Colon	Yes	Purulent	21	Wound infection	No
5	I12037620	64	M	2 days	No	Peptic ulcer	Duodenum	Yes	Purulent	25	RC	no
6	I12036051	25	M	2 days	No	Peptic ulcer	Duodenum	Yes	Purulent	20	Nil	no
7	I12034597	41	M	2 days	No	Trauma	Ileum	Yes	Purulent	20	RC	no
8	I12034447	60	M	<1 day	Yes	Peptic ulcer	Duodenum	Yes	Clear	22	Nil	no
9	I13002535	29	F	2 days	No	Appendicitis	Appendix	No	Purulent	15	Nil	no
10	I13001567	54	M	2days	No	Peptic ulcer	Stomach	No	Clear	13	Nil	no
11	I13001132	35	M	2 days	No	Appendicitis	Appendix	No	Purulent	10	Nil	no
12	I13000993	27	M	4 days	No	Non specific	Ileum	Yes	Faecal	26	RC, burst abdomen,post op ventilator support	no
13	I13000515	83	M	3 days	No	malignancy	Appendix	No	Purulent	19	Prolonged ileus	no
14	I13000495	27	M	4 days	No	Non specific	Colon	Yes	Faecal	22	Wound infection, sepsis. Stoma.	No
15	I13000141	15	M	2 days	Yes	Trauma	Jejunum	Yes	Purulent	27	RC, renal failure	no
16	I13005620	24	M	<1 day	No	trauma	Jejunum	Yes	Clear	10	Nil	no
17	I13005628	65	M	<1 day	No	Trauma	Jejunum	Yes	Clear	15	RC	No
18	I13005345	48	M	2 days	No	Peptic ulcer	Stomach	Yes	Clear	14	Nil	no
19	I13004366	67	M	2 days	No	Nonspecific	Jejunum	Yes	Faecal	31	wound infection,stoma	No
20	I13009677	25	M	2days	No	Appendicitis	Appendix	No	Purulent	10	Nil	no
21	I13009506	75	M	3 days	No	Appendicitis	Appendix	Yes	Purulent	15	Wound infection	no
22	I13009064	75	M	2 days	Yes	Non specific	Colon :Rectum	No	Purulent	22	Prolonged ileus	no
23	I13006998	65	M	3 days	Yes	Trauma	Colon : Caecum	Yes	Faecal	34		Yes : septic shock
24	I13007289	55	M	2 days	No	Peptic ulcer	Duodenum	No	Clear	13	Nil	no
25	I13006696	68	F	3 days	Yes	Obstruction	Ileum	Yes	Clear	31		Yes : septic shock
26	I13013380	55	M	3 days	No	Enteric	Ileum	Yes	Purulent	25	Ventilator support, RC, wound infection	no
27	I13012359	39	M	4 days	Yes	Ischaemia	Jejunum	Yes	Clear	22	Nil	No
28	I13016999	46	M	2 days	No	Appendicitis	Appendix	No	Purulent	10	Nil	no

29	I13016889	69	F	3 days	No	Appendicitis	Appendix	No	Purulent	20	Nil	no
30	I13015369	40	M	4 days	Yes	Ischaemia	Ileum	Yes	Purulent	27	wound infection,burst abdomen	no
31	I13014990	21	M	<1 day	No	Appendicitis	Appendix	No	Purulent	10	Nil	no
32	I13014977	53	F	2 days	No	GIST	Duodenum	No	Clear	18	Anastomotic leak	no
33	I13014213	46	F	2 days	No	Appendicitis	Appendix	No	Purulent	10	Nil	No
34	I13018323	67	F	4 days	No	Appendicitis	Appendix	No	Purulent	20	Fistula	No
35	I13017206	43	M	<1 day	No	Trauma	Ileum	Yes	Clear	10	Nil	No
36	I13016971	36	M	3 days	No	Inflammatory bowel disease	Colon	Yes	Purulent	16	Stoma	No
37	I13022458	25	M	4 days	No	Tuberculous	Jejunum	Yes	Purulent	20	Post op ventilator support	no
38	I13022432	19	M	2 days	No	Appendicitis	Appendix	No	Purulent	10	Nil	no
39	I13021814	46	F	2 days	No	Appendicitis	Appendix	No	Purulent	15	Nil	No
40	I13026245	45	M	4 days	No	Malignancy	Colon	Yes	Faecal	26	Prolonged ileus, wound infection	no
41	I13025855	31	M	<1 day	No	Enteric	Ileum	Yes	Purulent	16	Wound infection	no
42	I13024284	53	M	2 days	No	Peptic ulcer	Duodenum	No	Clear	13	Nil	no
43	I13024053	59	M	3 days	Yes	Cholecystitis	Gall bladder	No	Purulent	20	Wound infection,bile leak,RC	no
44	I13032366	17	M	2days	No	Appendicitis	Appendix	No	Purulent	10	Nil	No
45	I13030674	23	F	2 days	No	Appendicitis	Appendix	No	Purulent	15	Nil	No
46	I13034278	32	M	2 days	No	Peptic ulcer	Duodenum	Yes	Clear	14	Nil	no
47	I13033842	62	F	4 days	No	Inflammatory bowel disease	Colon	Yes	Purulent	26	Renal failure,post op ventilation	no
48	I13033245	32	M	< 1 day	No	Trauma	Duodenum	Yes	Clear	10	Nil	no
49	I13032685	53	M	4 days	No	Nonspecific	Ileum	Yes	Purulent	25	RC	No
50	I13022554	46	M	2 days	No	Trauma	Ileum	Yes	Purulent	20	RC	no

RC – RESPIRATORY COMPLICATIONS